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The Pathophysiology of High Sustained $+G_z$ Acceleration, Limitation to Air Combat Manoeuvering and the Use of Centrifuges in Performance Training

NORTH ATLANTIC TREATY ORGANIZATION

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THE PATHOPHYSIOLOGY OF HIGH SUSTAINED $+G_z$ ACCELERATION, LIMITATION TO AIR COMBAT MANOEUVERING AND THE USE OF CENTRIFUGES IN PERFORMANCE TRAINING

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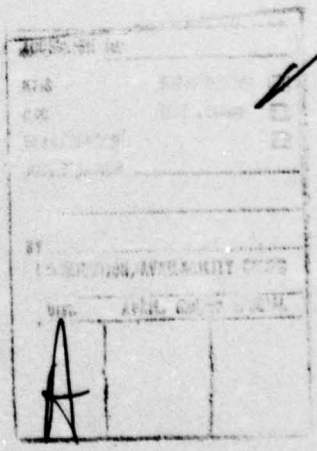

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PREFACE

This symposium was organized to present and discuss relevant information on the pilot effects of maneuvering acceleration provided by new systems such as the F-15, F-16 and F-14. High levels of acceleration can be sustained for relatively long periods of time in these systems. Measurements of acceleration profiles developed in current aircraft (F-4) show that pilots used brief peak accelerations of up to +8-9 G_z to gain advantage. They frequently remained at acceleration levels above +4-5 G_z for many seconds with the high peaks superimposed on the lower longer duration loads. Among other things, the duration of high peak accelerations was previously limited by available energy, a situation which does not prevail with new fighter aircraft such as those mentioned above.

There is not enough operational experience at this time to say how a fighter pilot will use the increased maneuvering energy available to him with new systems. Engagements of aircraft with similar high performance capabilities may well involve extension of present tactics or development of new tactics which will involve sustaining very high peak accelerations for longer times. The "baseline" level of acceleration on which these peaks are superimposed may also be at a higher level than presently used. Although we cannot say how the fighter pilot will use this new "reserve" maneuvering energy, history would lead one to predict that he will find a way to turn the maximum performance of the aircraft to his advantage. For this reason, there is need for information on the risk involved in exposure to the high-sustained +G_z environment and on methods to enhance the pilot's ability to effectively use this environment.

The organization and execution of this symposium was designed to specifically address a series of preconceived pertinent operationally oriented questions on the effects of high sustained +G_z accelerations and on the use of centrifuges in training pilots to perform more effectively during acceleration. These questions were submitted to the speakers in advance of the meeting. They were addressed by speakers during their presentations and were then reviewed and discussed in a round-table forum at the end of the session. The round-table discussion focused specific attention on the operationally significant conclusions which can be drawn on the questions with our present state of knowledge. Because of its relevance, this session was transcribed, edited and included in the proceedings.

Following are the questions which were addressed in this symposium:

1. What is the pathophysiologic risk to a tactical fighter pilot exposed to aerial combat accelerations, with duration above +4G_z in excess of 60 seconds and peaks up to +8-10G_z, in the case of:
   a. Single exposures?
   b. Several exposures per day each day for several days?
   c. Repeated exposures that might be encountered in a career as a fighter pilot?
2. What are the physiologic factors that provide for practical performance during high G maneuvers and what are the limits of performance enhancement they provide?
3. What is the quantitative relationship between body position (including back angle) and enhanced tactical crew performance during high acceleration? What studies are needed to provide definitive answers to this question?
4. What general and specific recommendations can be made on the usefulness of centrifuge training to enhance fighter pilot performance? What training regimes and methodology are optimal?
5. What are the problems in cockpit design for control, display, visibility and mobility that must be solved to take nominal and/or maximum advantage of increased back angle during high accelerations?
6. What is the effect of physical conditioning on pilot performance during high +G_z acceleration?
7. What are the practical safe limits in terms of pilot performance and fatigue to repeated low acceleration missions during a single day?

The risk of significant cardiovascular change to a tactical fighter pilot from single exposures to aerial combat accelerations such as those postulated for new high performance aircraft is believed to be low, based on human centrifuge studies and one previous operational experience. Simulations of profiles of accelerations postulated for air combat maneuvering in the F-15 and F-16 were tolerable but fatiguing, uncomfortable and produced hemorrhage in the skin of dependent parts of the body. Cardiac arrhythmias frequently occurred late in the exposure; their significance is not known but they appear to be, in part, associated with fatigue. Electrocardiography and measurement of biochemical indicators of damaged tissue do not reveal evidence to show that man sustains the injury to the heart observed in miniature swine, which were used as animal models to study the effects of high sustained acceleration. However, there is some uncertainty involved in this conclusion since only indirect evidence on human cardiac effects can be obtained. In miniature swine, high sustained $+G_z$ accelerations produce hemorrhage in the heart muscle and under the lining of the cardiac chambers. Some microscopic damage to muscle fibers was observed. These changes require both high level and long duration of acceleration. The pig is believed to be a relatively good animal model of man as regards this problem. However, experts believe he is a conservative model, i.e., more apt to show effects than man. It is clear that man is operating near his limits of endurance at accelerations above +6 G_z in conventional cockpits. Protective equipment and straining are required and interruption or failure of these mechanisms will lead to loss of vision and probably unconsciousness. Concomitant environmental stressors such as cockpit heat, hypoxia and psychological factors may be additive to the high sustained acceleration effects. Good environmental conditioning should not be compromised in such aircraft.

It was the consensus of most speakers that the risk of several exposures per day for several days to the high-sustained G environment is also relatively low, provided excessive fatigue is not incurred. There is less certainty in this conclusion since the experimental studies with humans which would provide data on this question have not been completed.

The risk of cardiac damage to a fighter pilot from repeated exposures over a flying career still more difficult to assess. If a damage pattern similar to that seen in the miniature swine occurs in man, one might project a cumulative effect which could eventually be significant. The limited studies which have been done to date do not suggest that operational pilots have suffered measured ill effects. More research is urgently needed to examine this question.

The practical methods for enhancing G tolerance and the order of enhancement provided are given below. These are average figures; individual pilot variability is large.

	Increased Blackout Threshold ($+G_z$)
1. Anti-G suit	1 - 1.5
2. M-1 or L-1 straining maneuver	1 - 2.0
3. Positive pressure breathing	0.75

The M-1/L-1 maneuver and positive pressure breathing are not used together. The tilt-back seat concept forms the other major physiologic method to improve tolerance. Results from the USAF School of Aerospace Medicine suggest that a program of physical conditioning, including weight lifting and running provides an improvement in $+G_z$ tolerance. Since there is significant variability in ability to withstand $+G_z$ forces, it would be desirable to use this as one criterion in the selection of pilots to fly high performance aircraft.

The first significant effect of increasing backward tilt of the seatback occurs at an angle of about 45°. The major physiologic advantage accrues at an angle of approximately 65°. Several innovative methods have been proposed to capture the hydrostatic advantage of tilting while maintaining an acceptable position for operating the aircraft. Definitive research is needed to quantitate the operational advantage (increased kill probability) of the tilt back seat approach.

Experienced fighter pilots strongly feel that centrifuge training improves their ability to effectively perform in the high G environment. All aircrew who have experienced it recommend centrifuge training for fighter pilots. They report that they can often achieve higher G levels without blackout after training. Perhaps more importantly, most pilots have never experienced exposure to high G for the longer durations now possible with modern fighters. Centrifuge training is a great asset to the pilot in learning to effectively perform the necessary straining maneuvers for these longer periods without blacking out or becoming excessively fatigued. For these reasons, it is recommended that all pilots flying air superiority weapon systems receive high G centrifuge training if resources permit. This training would be most valuable following completion of undergraduate pilot training, while the pilot is undergoing training in air combat maneuvering. It is probably not necessary for the pilot to repeat this training unless he is disassociated with the high G environment for a long period.

CHANGES IN CLINICAL CARDIOLOGIC MEASUREMENTS ASSOCIATED WITH HIGH $+G_z$ STRESS

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ABSTRACT

Because of reports of subendocardial hemorrhage and myofibrillar degeneration in animals exposed to sustained high G loads (> +6 G_z for 15 s or more), questions have been raised regarding the safety of exposing pilots and human subjects to the similar G-stress levels likely to be encountered in the new high-performance fighter aircraft. Non-invasive clinical cardiologic data, including ECGs, vectorcardiograms, systolic time intervals, and maximal treadmill stress tests, were obtained from 2 groups of subjects before and at several times after exposure to high-G stress. The group exposed to the greater G stress (3 40-s runs at 8 G and 2 40-s runs at 10 G, all in one day) developed moderate cutaneous petechiasis and had other minor physical findings after the G stress, but showed few significant changes in cardiologic data: serum total CPK and LDH levels rose, and preejection period shortened at 48 h poststress. The group exposed to the lesser G stress (one 100-s variable-G maneuver peaking twice at 8 G for 3 s, once a week for 3 weeks) had no symptoms following the G stress, but the vectorcardiograms revealed transient T-loop angle changes, and preejection period measured at 1 week poststress was significantly decreased. Because the serum enzyme changes were noncardiac in origin, and because the few other changes were not in a direction indicative of cardiac damage, we conclude that the G stresses imposed in these studies were not significantly injurious.

The mechanical strength and propulsive power of the current spawn of fighter aircraft (F-14, F-15, F-16, F-18) are so great as to potentially exclude the pilot from substantial portions of the maneuvering envelopes of these aircraft. Whereas aerial combat between fighters was in the past characterized by brief spikes of high (+6 to +9 G_z) load forces which rapidly dissipated maneuvering energy, the aerial engagements between our newer fighters and adversaries of like performance are expected to take place in an environment of sustained high G loading--+6 G_z or greater for 15 seconds or more. In such a combat environment, the winner may not so likely be determined by an advantage in aircraft performance as by the ability and motivation of the pilot to withstand the tremendous physiologic stress of the sustained G load. To doubt that fighter pilots will accept the challenge in this newly created arena of aerial combat is unreasonable; to assume they will engage with physiologic impunity in real or simulated aerial combat in the sustained high-G environment is, at this time, also unreasonable. The fact that a handful of well-trained subjects have recently ridden a human centrifuge to +9 G_z for 45 s with anti-G suit and straining maneuver (24), and at least one has sustained 14 G for 45 s in a 75° PALE (pelvis-and-legs-elevating) seat with anti-G suit and straining (1), proves only that a pilot can enter the sustained high-G environment, not that he will emerge unscathed.

What sorts of pathologic effects of exposure to sustained high-G stress can actually be seen? Most obvious by far are the cutaneous petechial hemorrhages which appear on the abdomen, back, buttocks, legs, feet, arms and hands of experimental subjects who have been vigorously straining to tolerate sustained high G forces (4, 27). These hemorrhages appear in areas of skin where the anti-G suit exerts no pressure, are sometimes confluent and sometimes seen as reddish-purple striae, but are painless and usually resolve completely within a few days. The petechiae almost always occur below heart level, and generally are most severe in the more dependent areas of the body, leading one to believe that high intravascular pressures are at least part of the mechanism of their production. Also seen after repeated high-G runs is edema of the feet and ankles, clearly resulting from prolonged high hydrostatic pressures in these dependent parts.

A set of fairly consistent consequences of high $+G_z$ stress, observable in humans monitored electrocardiographically during runs of the USAFSAM human centrifuge, are described by Shubrooks (25). He reports sinus tachycardias as high as 205, relative bradycardias as low as 75, junctional rhythms, multiform ventricular premature beats (VPBs), coupled VPBs and ventricular tachycardia, ST-segment depression, and T-wave amplitude changes, in subjects during exposure to +6.5 to +9.0 G_z for up to 45 s. In addition to the above, we have seen atrial premature beats, bigeminy, and postacceleration bradycardias as low as 30 in experimental subjects sustaining high $+G_z$ stress on the USAFSAM human centrifuge during the past 2 years. Subjects may also suffer a mild disorientation, lasting several hours to one day after the high-G stress. This symptom probably results from perstimulatory adaptation to the new motional environment, which the subject experiences as a temporary maladaptation upon his return to the 1-G environment. It is possible, however, that the disorientation is caused by minor G-induced damage to the otolith organs (17, 23), and that central nervous system compensation causes the symptoms to disappear. Other symptoms, inconsistently reported after high-G runs, are myalgia, pain in various joints, and pain over bony prominences that lay in contact with the seat.

While cutaneous petechiasis itself is of negligible concern, its consistent appearance as a result of high-G stress raises the question of whether organs other than the skin might consistently be hemorrhaging, with more serious pathophysiologic consequences. Of particular interest are the reports of cardiac hemorrhage in animals exposed to high G forces (2, 6, 7, 13, 14, 16, 18, 21). The left ventricle is the site of predilection for the subendocardial hemorrhage reported by most of these authors; ridges and other prominences appear especially likely to be affected. In several instances, hemorrhage into the ventricular conduction (Purkinje) system was observed (6, 7), and at least one instance of abnormal ventricular conduction (widened, notched QRS) has been associated with such lesions (Burton, to be

published). Not only subendocardial hemorrhage, but also various types of damage to cardiac myocytes have been seen in miniature swine exposed to sustained high $+G_z$ stress, as reported by Lindsey, et al. (20), and MacKenzie, et al. (21); among the conditions seen were myofibrillar degeneration or "contraction bands," and frank necrosis of myocytes. Certainly adding to our concern are the reports of the following investigators: Erickson, et al. (11, 12), who showed substantially decreased coronary blood flow during $+G_z$ stress in dogs; Chimoskey (9), who found G-induced decreases in coronary blood flow and T-wave flattening in ECGs of dogs with epicardial leads; and Forlini (unpublished data) who sampled venous blood from the great cardiac vein of dogs during $+G_z$ stress, and found changes in the ratio of lactate and pyruvate concentrations. These findings suggest that $+G_z$ stress produces acute ischemia and anaerobic metabolism in the myocardium.

We recognize the difficulties in extrapolating human pathophysiology from animal data, especially when the degree of psychological stress on the trained pilot or centrifuge rider is so different from that on even the best animal model. We also realize that the physical conditioning of pilots and human experimental subjects is vastly superior to that of the experimental animals used in G-stress studies. Nonetheless, it is established that hearts of both animals and humans that have died of hypovolemic shock (8, 22) or of overdose of catecholamines (15) can exhibit subendocardial hemorrhage and myofibrillar damage. The suggestion could reasonably be made, therefore, that humans exposed to high $+G_z$ stress, wherein poor venous return and high levels of circulating catecholamines are to be expected, are liable to suffer subendocardial hemorrhage and myofibrillar damage, perhaps as a result of myocardial hypoxia. Cognizant of this, Walter (personal communication, 1973) examined vectorcardiograms (VCGs) obtained from centrifuge subjects before and after their exposure to +9 G_z for 45 s, and in 2 of 5 subjects found T-loop angle changes, of small amplitude but consistently in the direction of ischemic change, persisting up to 48 h after the G stress. Forlini (unpublished data), expanding Walter's VCG study, examined a total of 7 experimental subjects exposed to sustained +8 or +9 G_z stress on 4 separate occasions each, and compared their poststress VCG responses with their own preexposure VCGs as well as the VCGs of 5 unstressed control subjects. In the ensuing statistical analysis, 2 comparisons resulted in significant mean differences at the $p < 0.05$ level when either the paired t-test or the signed ranks test was used. They were the prestress vs 24-h-poststress sagittal-plane T-loop angle, and the prestress vs 24-h-post-stress sagittal QRS-T-loop angle difference, the latter of course being dependent on the former. Considering that nearly 130 comparisons were made, we were not surprised to find 2 comparisons revealing differences significant at the $p < 0.05$ level, since one would expect 6 or 7 of 130 comparisons to reach 0.05-level significance, if the comparisions were made on independent random variables.

But because the vector shifts were significant, and possibly represented myocardial ischemia, they could not be ignored. We were obliged to consider that some form of damage to the heart, perhaps cardiac hemorrhage, might be resulting from exposure to sustained high $+G_z$ stress. If cardiac hemorrhage is occurring, permanent damage could result--specifically, hemorrhage into the Purkinje system could result in a heart block; hemorrhage into a papillary muscle could result in mitral valvular incompetence; and repeated hemorrhage into the subendocardium or myocardium could result in fibrosis with consequent decreased cardiac compliance and contractility. Therefore, to minimize the risk of cardiac damage to experimental subjects undergoing sustained high-G stress, subsequent high-G experimentation at USAFSAM was limited to levels of 7 G or less for one minute or less, pending the outcome of certain cardiologic studies which were to be done on subjects exposed to higher levels of G stress. Those higher levels of G stress were generated during two experiments conducted between July and December 1974, one in support of the Lightweight Fighter (LWF) program, and the other in support of the High Acceleration Cockpit (HAC) program. The purpose of this article is to report the results of the cardiologic studies obtained in conjunction with the LWF and HAC experiments.

METHODS

The less stressful of the two experiments was the LWF study, conducted by Burton, et al. (5), who examined the physiologic and subjective responses of 8 subjects exposed to simulated aerial combat maneuvering (SACM) G stress at 23°, 28°, and 40° seatback angles. Each subject in this study underwent a number of training runs at lower G levels before being exposed to the SACMs, which had average G levels of only 4.2 G for the 100-s durations, but rose twice to 8-G, 3-s peaks (Fig. 1).

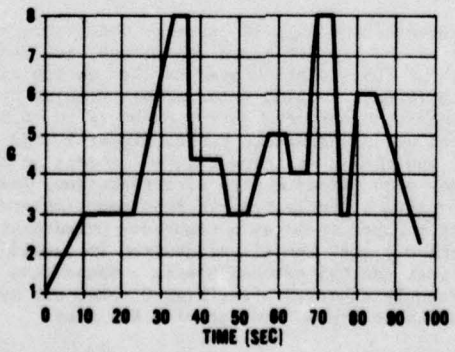

Figure 1. Variable-G profile (simulated aerial combat maneuver) used in the LWF study. Subjects were exposed to this stress once a week for 3 weeks.

The subjects rode the three SACMs at 1-week intervals. Before the training runs, each subject was given an Air Force Class II Flying physical examination; in addition, baseline VCGs, phonocardiograms, and systolic time intervals (STIs), as well as practice and baseline maximal treadmill stress tests, were obtained from each of the 8 subjects. After the final exposure to the SACM G stress, VCGs, phonocardiograms, and STIs were obtained immediately (within 1 hr), 24 hr later, and 1 week later, and treadmill tests were done 1 d later (Table I).

TABLE I. CLINICAL DATA COLLECTED ON THE LIGHTWEIGHT FIGHTER (LWF) STUDY

Test	1 week before training	1 h after last run	1 d after last run	1 week after last run
VCG	*	*	*	*
Phonocardiogram	*	*	*	*
STIs	*	*	*	*
Treadmill	*		*	

The more stressful of the two experiments, the HAC study, was conducted by Burns (3). He set out to subject 6 volunteers to a variable sequence of 5 sustained high-G exposures (Fig. 2): 8 G at a seatback angle of 13° from the vertical, 8 G at 45°, 8 G at 65°, 10 G at 45°, and 10 G at 65°, each exposure lasting approximately 40 s, and all exposures being completed within a 2-h period.

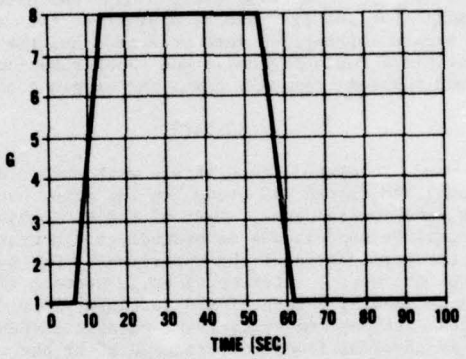

Figure 2. Constant-G profile used in the HAC study. Subjects were expected to sustain 5 of these at 8 or 10 G during one 2-h experiment.

Of the 6 subjects tested, none completed all 5 of the 40-s runs, and only 2 attempted all 5. Subjects terminated some or all of their runs early because of difficulty maintaining vision, excessive fatigue, or pain. (For that reason, we feel that the stress levels involved in this study represent a physiologic boundary, one which describes the limit of voluntary exposure of moderately trained, moderately motivated subjects.) Before the study started, each subject was given a Class II Flying physical examination. About one week before the G stress, the following tests were done (Table II): P-A and lateral chest roentgenograms, urinalysis, serum CPK, LDH, and GOT enzymes and CPK isoenzymes, phonocardiogram and STIs, and maximal treadmill stress test. One to 3 d before the centrifugation, the subject's heart and lungs were auscultated, a VCG and standard 12-lead ECG were obtained, and a 5-h continuous ECG was recorded with a Holter monitor. On the day of the acceleratory stress, as soon as the subject was deinstrumented from his esophageal balloon, radial artery cannula, ear oximeter, and ECG leads, a physician listened to his heart and lungs for poststress changes in his cardiopulmonary status. As rapidly in succession as possible, chest films, urinalysis, VCG and 12-lead ECG, phonocardiogram and STIs were obtained, and the subject was instrumented with the Holter monitor for the 5-h ECG recording. At approximately 24 h after the termination of the G stress, chest films, urinalysis, VCG and 12-lead ECG were again obtained, and blood was drawn for serum CPK, LDH, and GOT enzymes and CPK isoenzymes. Maximal treadmill stress testing was also done at this time. At 48 h after the experiment, VCG, ECG, phonocardiogram and STIs were done, and blood was drawn for serum enzymes for the last time.

The analysis of the VCGs, acquired with the modified Frank lead system, was done by four different methods. The first two involved hand digitization of the QRS and T loops from Polaroid photographs. The other two involved the averaging of 10 beats stored on magnetic tape (X, Y, and Z leads) and then the digital reconstructing of the QRS and T loops from the orthogonal lead data. In addition, the pertinent QRS and T-loop vectors were determined for each set of data by both the "half-area" method, wherein the vector dividing the T-loop areas into equal halves was selected, and the "maximum-length" method, wherein the vector with greatest magnitude was chosen. Paired t-tests were then used to compare the various parameters of the VCGs at the different times in relation to the G stress.

The STIs were analyzed in the conventional manner, with Weissler's regression equations being used to adjust Q-S_2, LVET, and PEP for heart rate (28). Paired t-tests were used for making comparisons between STIs obtained at the different times in the study.

TABLE II. CLINICAL DATA COLLECTED ON THE HIGH ACCLERATION COCKPIT (HAC) STUDY

Test	1 week before runs	1 to 3 d before runs	1 h after runs	24 h after runs	48 h after runs
Chest films	*		*	*	
Urinalysis	*		*	*	
Serum enzymes	*			*	*
Auscultation		*	*		
VCG		*	*	*	*
12-lead ECG		*	*	*	*
Holter monitor		*	*		
Treadmill	*			*	
Phonocardiogram	*		*		*
STIs	*		*		*

A modified Balke protocol (5% grade increase every 3 min) was used in the maximal treadmill stress test, and the baseline treadmill test was preceded by a practice test about 1 week prior. Treadmill time (endurance) and maximum oxygen consumption were measured, and the ECG recorded during and immediately after the test was scrutinized for arrhythmias and repolarization changes. Paired t-tests were performed to compare prerun and postrun treadmill times and oxygen consumption.

RESULTS

The subjects in the LWF study tolerated the G stress without incident and without obvious evidence of physiologic strain. The subjects in the HAC study, on the other hand, had a variety of symptoms which occurred during and after the experimental runs. Most of the HAC subjects perspired freely during the G stress--even though the centrifuge gondola was maintained at a comfortable temperature--and they all were physically exhausted at the termination of the experiment. One subject complained of a deep, dull pain in his left chest at about the second intercostal space between the midclavicular and anterior axillary line, radiating into the medial aspect of his left arm; this complaint resulted in termination of his exposure to the G stress, although no evidence of cardiac ischemia was present on the ECG monitor at the time, and no symptoms or physical findings persisted after the run. Another subject complained of mild pain of the left forearm and medial upper arm on the day following the G stress, but no evidence of physical damage was discernible. Of possible bearing in these two cases is the fact that an arterial cannula was placed in the left radial artery, and it was flushed vigorously every 15 minutes with heparinized saline to maintain patency of the arterial pressure-sensing line: possibly, retrograde distribution of the saline irritated the radial and brachial arteries, resulting in the arm pain. A third subject lost consciousness during one of the runs, terminating his participation; then he vomited in the treatment room when his radial arterial cannula was removed. We presume this subject's postrun symptoms were due to an emotionally triggered vagal reaction. All 6 of the HAC subjects had some degree of cutaneous petechiasis, 3 having an amount graded as moderate (10 - 50% of specified area of skin involved, but minimal confluence of lesions). The characteristic distribution of petechiae was over the lateral chest walls, the hips and lateral thighs, and the lumbar vertebral furrow. One subject reported that he had a painful right testis associated with a "bruised discoloration" for several days after his runs, but no evidence of this was found on physical examinations done immediately and one week after the runs.

All chest films and urinalyses of the HAC subjects were normal, and auscultation of the heart and lungs revealed no significant differences between prerun and postrun conditions.

No myocardial CPK isoenzyme (MB fraction) was found in serum taken from the HAC subjects before or 24 or 48 h after exposure to the G stress. The mean total serum CPK, LDH, and GOT obtained at the different times are presented in Table III. Although CPK and LDH rose significantly at 24 h and remained high at 48 h, it was not surprising to see the rises, since CPKs of 1000 and LDHs of 500 are not uncommon after strenuous muscular effort, and these subjects were applying substantial isometric effort during their straining to tolerate the G forces.

Table IV summarizes the results of the maximal stress testing on the treadmill. Neither the HAC nor the LWF subjects provided appreciable differences between preexposure and postexposure values for either treadmill time or maximum oxygen consumption. No electrocardiographic abnormalities were noted during any of the treadmill tests.

Holter monitoring of the HAC subjects revealed no significant abnormalities of rhythm either before or after the G stress.

The systolic time intervals before and after G stress are summarized in Table V. Since the postrun values of the PEP, PEPI and PEP/LVET ratios were generally lower rather than higher than the baseline values, and since the LVET and LVETI values remained essentially unchanged, it appears that at least no decrement in left ventricular performance resulted from the G stress. The reason for the statistical significance of the mean difference between the HAC subjects' prerun and 48-h-postrun PEPIs and between

the LWF subjects' prerun and one-week-postrun PEPIs and PEP/LVET ratios is presently not clear. Had more rigorous procedures been employed to insure during each examination a constant preload, afterload, and heart rate, there would probably have been less variability in the results than was observed.

TABLE III. HAC SUBJECTS' SERUM ENZYMES BEFORE AND AFTER G STRESS (IU)

	Before	24 h after	48 h after	Normal value
CPK				
Mean	106	186*	234*	< 105
S.E.M.	49	79	98	
LDH				
Mean	164	203**	202	< 295
S.E.M.	35	40	52	
GOT				
Mean	13.3	13.9	13.5	< 40
S.E.M.	4.0	3.5	4.2	

*One-tailed paired t-test shows $p < 0.05$ for a value so much larger than the preexposure value.

**$p < 0.005$ (1-tailed paired t-test).

TABLE IV. TREADMILL PERFORMANCE OF HAC AND LWF SUBJECTS BEFORE AND AFTER G STRESS

	Endurance (min)		Maximum O_2 consumption (ml/kg/min)	
	Before	After	Before	After
HAC subjects*				
Mean	14.60	14.80	41.40	40.10
S.E.M.	0.68	0.58	2.39	0.60
LWF subjects				
Mean	13.94	14.19	43.75	41.35
S.E.M.	0.60	0.68	1.20	1.53

*One of the HAC subjects was eliminated from the analysis--he complained that his "ankles hurt" during the postexposure treadmill test and terminated the test before reaching his maximum effort. Neither difference is statistically significant, even when this outlier is included.

When the subjects' VCGs were examined visually, no evidence of any pathologic condition resulting from the exposure to G stress was apparent, although one subject had a nonspecific terminal conduction delay and another had borderline increased voltage on all 4 examinations. The statistical analysis of the VCG data involved 1480 separate comparisons. Of these, 111 or 7.5% resulted in probablities of 0.05 or less. To get to the meat of the statistical results, the probabilities associated with the comparisons were tabulated so that rows of low probability could be identified in a matrix consisting of 185 rows (representing VCG parameters) and 8 columns (representing the 4 methods of analysis for each of the 2 subject groups). Any row containing 3 or 4 significant ($p < 0.05$) comparisons out of the 4 possible in either subject group was presumed to represent a parameter worthy of note because its values had changed in a consistent manner. The VCG parameters which emerged from this screening were 4 in number, all from the LWF study: the sagittal-plane T-loop angle, pre vs 1 h; the sagittal T angle, pre vs 24 h; the transverse T angle, pre vs 1 h; and the transverse QRS-T angle difference, pre vs 1 week. Obviously, the VCG changes of interest were in the T loops, suggesting that the repolarization process was affected by the G stress. This result is even more disturbing when one recalls that Forlini's study of VCG changes related to G stress also demonstrated significant changes in repolarization (sagittal T angle, pre vs 24 h; and sagittal QRS-T angle difference, pre vs 24 h). Table VI contains the mean T angles and the differences between the QRS and T angles for the subjects in the LWF, HAC, and Forlini experiments. Only 1 of the 4 types of VCG analysis is represented in the table--that in which photographs of the vector loops were digitized manually and a computer was used to determine those vectors which divided the respective loops into equal areas. These data show that, despite the commonality in

parameters showing statistically significant differences, the directions of the changes were not consistent; i.e., the sagittal-plane T angle of the LWF subjects decreased (statistically significantly at 1 h) after G stress, while that of the HAC subjects increased (but not significantly), and that of Forlini's subjects remained about the same (even though a significant increase was registered at 24 h). A similar picture of inconsistent variation emerged when the results of the other 3 VCG analysis methods were studied. When the results of the statistical analysis were known, we reviewed all of the photographs of the VCG loops to see if we could recognize the changes which the statistics indicated were there. The changes were present as indicated, recognizable in retrospect.

TABLE V. SYSTOLIC TIME INTERVALS (MILLISECONDS) OF HAC AND LWF SUBJECTS BEFORE AND AFTER G STRESS (MEANS ± S.E.M.)

HAC Subjects

	Before	1 h after	24 h after	48 h after
QS_2	403.3 ± 4.4	401.3 ± 5.0	402.5 ± 5.5	403.7 ± 9.2
QS_2I	552.0 ± 19.6	534.3 ± 7.4	522.8 ± 4.4	528.7 ± 4.7
LVET	310.5 ± 6.0	304.5 ± 4.6	313.0 ± 5.5	320.0 ± 8.9
LVETI	430.7 ± 17.1	412.0 ± 4.9	410.3 ± 3.4	421.0 ± 3.4
PEP	92.8 ± 3.4	96.8 ± 5.0	89.5 ± 0.3	83.7 ± 2.0
PEPI	121.2 ± 4.5	122.2 ± 5.6	112.5 ± 1.5	107.3 ± 2.8*
PEP/LVET	0.300 ± 0.016	0.319 ± 0.019	0.284 ± 0.004	0.262 ± 0.010

LWF Subjects

	Before	1 h after	24 h after	1 week after
QS_2	390.1 ± 9.8	382.4 ± 6.1	375.0 ± 8.7	392.0 ± 8.4
QS_2I	520.1 ± 5.6	517.7 ± 3.6	512.0 ± 6.8	515.1 ± 6.5
LVET	294.9 ± 7.8	295.1 ± 5.4	287.0 ± 9.5	305.7 ± 7.9
LVETI	400.1 ± 4.9	404.6 ± 3.4	397.9 ± 7.3	405.4 ± 6.2
PEP	95.3 ± 3.4	87.3 ± 3.2	88.0 ± 2.7	86.3 ± 2.2
PEPI	120.0 ± 2.9	113.0 ± 3.2	114.2 ± 3.2	109.7 ± 2.3*
PEP/LVET	0.323 ± 0.012	0.296 ± 0.013	0.310 ± 0.017	0.283 ± 0.010*

*$p < 0.05$ for so great a difference from prestress value (2-tailed paired t-test).

DISCUSSION

Besides cutaneous petechial hemorrhage and ecchymosis, the subjects in the HAC study presented very little in the way of sustained physical symptoms or signs of physical damage. The rises in serum CPK and LDH were certainly due to the muscular activity associated with the straining maneuvers (M-1 or L-1) used by the subjects to increase their G tolerance; this is consistent with Burns' (4) observation that straining to tolerate G forces increases serum levels of skeletal-muscle (MM) CPK isoenzyme, and with the data of Schwertner and Krutz (26) who reported elevated levels of serum CPK-MM, GOT, and LDH-P in subjects exposed to sustained acceleratory stress of +7 G_z or greater. The absence of pathologic changes in the auscultatory findings, chest films, ECGs, treadmill performance and maximum oxygen consumption, and the lack of consistent evidence for VCG changes, mitigate strongly against a conclusion that cardiac damage was inflicted on the HAC subjects by their exposure to sustained high acceleratory stress. The significant changes in those subjects' STIs (PEPIs) at 48 h after the stress are not worrisome: as already mentioned, the changes are at least not indicative of decreased cardiac performance.

The subjects in the LWF study, although exposed to far less acceleratory stress, gave a bit more in the way of statistically significant changes in measurements of interest. The changes in their STIs (PEPI and PEP/LVET ratio) were not alarming, since, as with the HAC subjects, the changes were not in the direction of decreased cardiac performance. The post-G-stress changes in T angle and QRS-T angle difference in the LWF subjects' sagittal- and transverse-plane VCGs do not, we believe, signify that serious cardiac repolarization abnormalities resulted from the G stress. We say this considering that, although T-wave changes are notoriously nonspecific, they are commonly observed in healthy people exposed to mild gravitational stress such as occurs with use of the tilt table or low-level $+G_z$ centrifugation. Such changes are, according to Lamb (19), "so routine that in evaluating large numbers of subjects undergoing [tilt-table tests] our laboratory ignores them unless they have some unusual features." The LWF subjects' poststress T loops certainly had no unusual features, the changes that did occur being so subtle as to be barely perceptible on visual inspection. Reviewing the etiology of T-wave changes during mild $+G_z$ stress, Cohen and Brown (10) concluded that increased sympathetic tone was most likely responsible for T-wave changes observed for up to 30 s after termination of acceleration. It is debatable whether the LWF subjects' T-loop changes, if they were of sympathetic origin, should have persisted for hours to days after termination of the G stress; but in view of the unusually large magnitude of the

gravitational stress to which these subjects were exposed, the persistence of the T-loop changes beyond the immediate time of the stress should not come as a complete surprise. The absence of significant changes in the HAC subjects' T loops, and the opposite direction of the T-loop changes in Forlini's study, in the face of higher G-stress levels in the HAC and Forlini studies than that in the LWF study, attest in a reassuring way to an innocuous variability rather than an ominous consistency of T-loop changes following G stress.

TABLE VI. T ANGLES AND QRS-T ANGLE DIFFERENCES (DEGREES) BEFORE AND AFTER G STRESS (PHOTOGRAPHIC HALF-AREA METHOD; MEANS ± S.E.M.)

	Frontal Plane		Sagittal Plane		Transverse Plane	
	T Angle	QRS-T Angle Difference	T Angle	QRS-T Angle Difference	T Angle	QRS-T Angle Difference
LWF Subjects (N = 8 or less)						
Before stress	32.4±2.8	9.8±2.5	43.1±4.8	76.2±7.8	34.3±2.9	57.9±7.2
1 h after	31.2±4.7	11.7±3.1	33.1±3.8*	87.2±7.8*	42.9±3.9*	70.6±7.9*
24 h after	33.1±4.7	9.0±2.8	35.3±2.9	85.8±6.8	42.3±3.5*	68.9±8.7
1 week after	32.9±5.2	8.7±2.3	39.0±3.7	85.4±9.6	42.5±4.5*	71.3±9.3*
HAC Subjects (N = 6 or less)						
Before stress	35.7±6.7	8.6±7.1	34.4±6.2	97.8±10.1	47.6±8.9	86.2±6.8
1 hr after	34.2±3.3	10.6±4.9	40.4±4.3	100.5±8.4	40.6±3.9	80.7±8.4
24 h after	39.5±3.4	10.9±5.6	42.4±6.0	91.7±11.2	41.7±6.0	81.9±11.0
48 h after	23.5±8.7	26.0±13.2	42.2±5.1	94.3±9.5	41.4±5.3	81.9±11.2
Forlini's Subjects (N = 7 or less)						
Before stress	32.1±4.0	17.6±4.6	28.4±5.2	106.1±6.4	42.2±5.0	86.3±9.3
1 h after	30.7±4.0	18.0±5.1	28.7±6.2	104.0±9.0	44.0±5.4	87.2±9.8
24 h after	33.9±4.6	18.5±5.0	30.7±5.6*	104.7±7.4*	43.3±5.5	91.2±9.1
48 h after	28.5±5.2	16.8±6.2	28.3±4.8	109.8±8.4	40.5±6.8	82.1±14.0
6 days after	29.3±2.9	19.1±6.9	23.9±4.3	112.3±7.0	47.6±6.9	93.2±10.8

*$p < 0.05$ for so great a difference from prestress value (2-tailed paired t-test).

In summary, then, we are unable to provide clear evidence of compromised cardiac performance resulting from high levels of G stress, based on the noninvasive measurements at our disposal. One could argue that the techniques used in this study are too insensitive to determine the presence of relatively small amounts of cardiac damage. This is probably true. We cannot justify at this time, however, the use of more sensitive invasive methods, all of which carry, in our opinion, a greater risk for the subject than does the exposure to sustained high-G stress of the type which we foresee as important in the next 20 years. And even though we were hampered somewhat in these studies by having to use data obtained by "piggy-backing" on others' experiments, we certainly cannot justify on moral grounds the conduct of a study dedicated solely to obtaining data on the pathologic effects of G stress on humans. We are at the present time continuing to collect data from animal models, primarily the miniature swine, in hopes of establishing beyond question the cause or causes of subendocardial hemorrhage and myofibrillar degeneration in animals exposed to sustained high-G stress. When the cause is firmly established, we shall be in a better position to extrapolate the results of the animal studies to the human situation, and can perhaps offer some form of cardiac risk assessment for pilots of high-performance fighter aircraft. Until that time, however, our recommendations are the following:

1. Continue to do high-G human experiments determined to be necessary because of operational requirements, limiting exposures to those G levels having definite operational significance.

2. Maintain adequate medical monitoring and treatment capability during all such experiments.

3. Collect appropriate cardiovascular data, such as VCGs, echocardiograms, phonocardiograms, and serum enzymes, before and after imposing G stresses of greater magnitude and/or duration than those for which data have previously been collected and analyzed.

4. Obtain whatever inflight and postflight physiologic and pathologic data possible from aircrew engaging in aerial combat maneuvering in the newer high-performance aircraft.

5. Examine for evidence of cardiac strain those aircrew accumulating substantial time in the sustained high-G environment.

6. Continue to collect data from experimental animals to further define any pathologic consequences of exposure to sustained high-G stress.

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DISCUSSION

McARTHUR (Canada): Were the subjects fighter pilots previously experienced in exposure to operational high G loadings?

GILLINGHAM: No. These subjects were members of the USAFSAM Acceleration Stress Panel, a group of 30 volunteer airmen and officers who ride the human centrifuge at least once a month, but generally have no actual flying experience.

BLACKBURN (United States): What rate of G onset was used in HAC and LWF studies on pigs and humans? What seatback angles were used in these studies?

GILLINGHAM: 1.0 G/sec. In the LWF study, 23^{o}, 28^{o}, and 40^{o} seatback angles were used. In the HAC study, 10 G was applied at 45^{o} and 65^{o} seatback angles, and 8 G was applied at 13^{o}, 45^{o}, and 65^{o} angles.

PAOLUCCI (Italy): How many Gs are necessary to have catecholamine secretion? What was the catecholamine output in these experiments? What is the time relation between acceleration and catecholamine release?

GILLINGHAM: We have as yet been unable to validate our measurements of serum catecholamine levels obtained in conjunction with G stress. I am confident, however, that in experimental subjects exposed to unfamiliar G stress levels catecholamines are released well prior to the onset of acceleration and continue to be released during the G stress.

SEM-JACOBSEN (Norway): Are you attempting to get an operational sample, taking in the pilots (10%) with lower stress tolerance to get the Gaussian distribution of man's (pilots') tolerance?

GILLINGHAM: We are currently involved in a study to determine the age stratified distribution of relaxed G tolerance in U.S. Air Force pilots. We have no plans at present to determine formally the G tolerance distribution of anti-G-suited, straining pilots, but experience tells us that pilots unable to tolerate $+7G_z$ for 15 seconds would definitely be in the bottom 10% of such a distribution.

KAISER (Denmark): Do you consider the residual volume to be able to reflect possible damage to the heart?

GILLINGHAM: I would be surprised if noninvasive residual volume measurements would reveal pathologic changes of the type which we are addressing, but we nevertheless have included echocardiography in the battery of cardiac examinations we are currently using in the continuation of the "high G pathology" study.

VENTRICULAR PATHOLOGY IN SWINE AT HIGH SUSTAINED $+G_z$

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SUMMARY: Study of miniature swine has shown two distinct types of cardiac pathology as the result to exposure to HSG_z. Grossly visible endocardial hemorrhage of varying degrees of severity occur consistently. In severe cases damage to Purkinje fibers is adequate to explain some of the ECG changes that have been found. A stress myocardiopathy is also found characterized by randomly distributed single or grouped, degenerate and dead muscle fibers surrounded by normal appearing fibers. Electron microscopically the lesion is characterized by profound changes in the contractile myofibrils known as myofibrillar degeneration. These changes have also been found in Purkinje fibers. It appears that the subendocardial hemorrhage is related to the combination of tachycardia, strong contractions (positive inotropism), and a hypovolemic ventricle. The stress myocardiopathy has a distinctly different and complex etiology. The ultrastructural lesions are not indicative of a primary hypoxic insult although hypoxia undoubtedly contributes.

The requirement for man to function in the high sustained G (HSG) environment has involved extensive investigation to insure that he can tolerate this environment without injury. It is generally recognized that the heart is the most vulnerable organ to HSG_z. The use of adult miniature swine as an animal model (1) has shown extensive cardiac pathology in swine sustaining HSG_z stress (2). The studies reported here were undertaken to further delineate the parameters and mechanisms responsible for the heart pathology found in pigs sustaining HSG_z.

MATERIALS AND METHODS

In this series of experiments, 17 adult female miniature swine were used. They were exposed to HSG_z on the animal end of the USAFSAM human centrifuge, by methods previously reported (1), to the schedule in table I. Propranolol or atropine was given I.V. after the pigs were restrained in the couch immediately prior to centrifugation. All rides were of 90 sec. duration. Twenty-four hours later they were necropsied. Slices of myocardium, .5 mm thick and 1.5-2 cm long, were removed from the left papillary muscle and the endocardial surface of the left and right ventricles. The slices were fixed by immersion or by perfusion via the thoracic aorta with 2% glutaraldehyde, divided into pieces 0.5 cm square, post-fixed in osmium tetroxide, embedded in Epon, cut on the ultramicrotome at 1μ, and studied by light microscopy. Areas were selected for thin sectioning and studied with an electron microscope. Tissues taken for paraffin embedding were processed by standard methods. Special stains were used as needed.

RESULTS

A summary of the results of the gross pathology experiments is given in table I. The only gross lesions noted were endocardial hemorrhages. These were graded 1 to 4 in severity: 1 indicated a barely visible hemorrhage within an area of less than 1 sq cm; 4, the most severe hemorrhage, involved extensive areas of wall or papillary muscle. The hemorrhage usually occurred in the most exposed prominences of chordae tendineae and papillary muscles. The hemorrhages were found in the left ventricle exclusively except in a pig receiving atropine and 9 G_z. Propranolol at 0.5 mg/kg completely protected the heart from hemorrhage, while 0.25 mg/kg partially protected it.

Light Microscopy

The hemorrhage occurred immediately beneath the endocardium, often surrounding and isolating the numerous Purkinje fibers found there. In severe cases the hemorrhage extended several mm into the myocardium, and Purkinje fibers were ruptured and contained RBCs. There was little inflammatory response at 24 hours.

Another lesion frequently found was myofibrillar degeneration (3), often called "contraction bands." These are areas of hypercontraction characterized by loss of the normal striated appearance of myocardial cells and formation of dense fuchsinophilic bands of condensed contractile proteins; the area between these bands is roughly granular and less dense than normal. These changes involved both single myocytes and large areas containing many myocytes. They most often affected cells immediately beneath the endocardium but were also found in deeper muscle cells in both the papillary muscles and ventricle walls. Unlike the hemorrhages, myofibrillar degeneration was found frequently in the right papillary muscle and occasionally in the right ventricle wall.

Necrosis of myocytes was frequently found on 1-μ plastic sections but could be seen on paraffin-embedded sections in only the most severe cases In the less affected animals the necrotic cells occurred singly or in groups of 2 or 3, surrounded by normal appearing cells. In those animals receiving atropine

and in the pig receiving 3, 7, and 9 G_z, plaques of necrosis measuring .5-1 mm were found in the right and left papillary muscles and left ventricle wall. In the severe cases there was an inflammatory response, but single necrotic cells did not seem to have elicited a response at 24 hours. Whether this was due to cell death considerably after the centrifugation or to some other factor could not be determined.

A score of 1-4 was devised to quantify these lesions: 1, for minimal change found in more than 2 or 3 cells; 2, for more severe changes affecting many individual cells; 3, when many cells or areas of 1 mm or more were involved; and 4, for a necrotic lesion large enough to be seen grossly - - this score was never used.

Electron Microscopy

One intriguing character of all electron microscopic changes seen was severely affected cells next to completely normal appearing cells. Changes seen were irregular distention of sarcoplasmic reticulum, intercellular edema, myelin figure formation, changes in nuclear chromatin distribution, pooling of mitochondria, severe morphologic changes in mitochondria, contraction band formation, thickening of Z disks, and condensation and precipitation of contractile proteins. In necrotic cells mitochondria contained crystals morphologically compatible with calcium phosphates. Only in one pig were fat vacuoles present. Glycogen was plentiful. No difference other than the degree of injury was seen between stressed pigs receiving propranolol, atropine, or no treatment.

TABLE I: Summary of results

Stress	Number of pigs	Hemorrhage score (a)	Myofibrillar degeneration	Necrosis of myocytes
Control	1	0	0	0
1 G control	2	0	0	0
9 G_z .25 mg/kg propranolol	1	(b)	1	0
9 G_z .5 mg/kg propranolol	3	0	2, 3, 3	(c)
9 G_z 4 mg atropine	2	7, 10	2, 2	2, 3
9 G_z then 9 G_z 4 mg atropine	1	4	2	3
15 G_z then 15 G_z 4 mg atropine	1	8	1	1
15 G_z	1	0	1	2
9 G_z	2	3, 2	1, 2	2, 2
9 G 65°	2	6, 1	1, 1	2, 0
3 G, 7 G, 9 G	1	4	2	3

(a) Hemorrhage score is the sum of the severity of the left papillary muscles, left ventricular wall, and right ventricle. The most severe score is 12.
(b) Hemorrhages not seen grossly but minimal hemorrhages found microscopically.
(c) One single cell found necrotic.

DISCUSSION

The endocardial hemorrhages seem to be associated with heart rate, strength of contraction, and the ventricular volume at the end of systole. At 15 G_z there is a precipitous bradycardia and no hemorrhage occurs. At 9 G_z the bradycardia also occurs but not to the extent of at 15 G_z, and moderate hemorrhages occur. Pretreatment with atropine blocks this bradycardia, and hemorrhages are severe. Propranolol, a beta blocking agent, completely protects the heart from hemorrhages. The distribution of the hemorrhages on the ridges and prominences of the endocardium tends to support the theory that they are traumatic in origin.

The stress myocardiopathy, although often qualitatively and quantitatively associated with hemorrhage, is a distinct lesion with a different and complex etiology. Heart rate and strength of contraction contribute to the severity of the lesion as demonstrated by the atropinized pigs. The high G bradycardia at 15 G_z did not protect the myocardium from degenerative changes, and propranolol actually increased the severity of the myofibrillar degeneration although necrosis did not occur. The most severe lesions were seen in the 9 G_z atropinized animals and in the untreated pig receiving 3, 7, and 9 G_z exposure. The stress myocardiopathy is probably multicausal with hypoxia and ischemia playing only a contributory role. Morphologically, the ultrastructural lesion most resembles catecholamine toxicity (4) and is distinctly different from that described for ischemia (5). There are also features in common with a wide variety of etiologies including electrical shock, "stone heart syndrome", Mg and K deficiencies, and hemorrhagic shock, to name a few.

The significance of these findings to man is unknown; it would be a mistake to apply them without qualification. For instance, in a physical conditioning or exercise sense, these pigs have not had to exert themselves to forage or engage in other survival activities. Also, the centrifugation with its attendant handling and restraint, is a most unnatural, and stressful circumstance for a pig. Stress alone has produced fatal myocardiopathy in swine (7). On the other hand, to completely ignore these findings would be an even greater error. Pigs are becoming an increasingly important model for man

because of the similarities in the cardiovascular system (7). Pigs have also been shown to be similar to man in the cardiac lesions of hemorrhagic shock (8). An understanding of the pluricausal nature of myocardiopathies is necessary to understand the full implication of these findings for man (6).

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The animals involved in this study were procured, maintained, and used in accordance with the Animal Welfare Act of 1970 and the Guide for the Care and Use of Laboratory Animals prepared by the Institute of Laboratory Animal Resources — National Research Council.

DISCUSSION

VOGE (United States) Being that the pig is different from the human (human upright, swine on all four), wouldn't this explain a great deal of the pathology found?

MACKENZIE The pig, as are all animal models, is a compromise and not identical to man in all respects. I do not believe this postural effect is a major factor in producing the observed pathology, nor can it be readily quantitated.

UNTERHARNSCHEIDT (United States) Would you comment on the mechanism of the hemorrhages in the heart? How many minutes after exposure did they occur? Did you use other animal species?

MACKENZIE Hemorrhages, being associated with tachycardia and positive inotropism could be expected to be traumatic in origin. We have seen broken capillaries and venules in electron micrographs. The endothelial cells were normal, which would support this contention. We have not used other animals, but hemorrhage has been reported in other species.

COWAN (United States) The gross photograph appears to have hemorrhage in the aortic valve and base of the aorta as well. Is this true, or are hemorrhage confined solely to the endocardium of the ventricle and papillary muscles? Do the areas of muscle degeneration demonstrated by electron microscopy coincide with areas of loss of fibers for eosin affinity, the earliest changes normally demonstrated by H and E stains?

MACKENZIE The hemorrhage did not involve the valves or major vessel walls. To the second question, no. In the two areas upon which we have done plastic sections for light and electron microscopy the number of affected muscle fibers found are more numerous and more easily seen than on parafin embedded H and E sections.

CHRISTIE (United Kingdom) Concerning the phenomenon of myofibrillar degeneration, what controls were carried out to ensure an association between high G effects and myofibrillar degeneration rather than the other stress features of the experiment?

MACKENZIE Control runs at $1G_z$ were carried out and animals were sedated prior to necropsy.

KAISER (Denmark) In order to protect the myofibers during the onset of each contraction would it be possible in animal experiments to increase the heart rate and thereby decrease the stroke-volume, for instance by pacing?

MACKENZIE We have paced a few hearts and found that increased heart rate is associated with increased injury.

PSYCHO-PHYSIOLOGICAL AND PHYSIO-CHEMICAL ASSESSMENT OF ACCELERATION INDUCED CHANGES IN HUMANS POSITIONED IN VARIOUS SEATBACK ANGLE CONFIGURATIONS

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INTRODUCTION:

A series of high-G tests were conducted at the Naval Air Development Center, Warminster, Pennsylvania, using the MACT (Multi-Posture Adjustable Centrifuge Test) seat. The rationale of this test series was to demonstrate an increase in human tolerance to sustained acceleration by employing several seat configurations.

There is still much discussion about the immediate and long range effects of "high-G" on pilots/subjects, [1,2] e.g. endocardial hemorrhage. Considering these questions, several psycho-physiological measurements were made during this test series. We will report here only those tests in which G_z accelerations were applied. (Sessions 1, 2, 3 and 4).

MATERIALS AND METHODS:

Ten subjects, Naval active duty personnel between the ages of 20 and 44 yr (mean 29.9 yr, mode 20-25 yr) with various body builds and G experience (Figure 1), took part in this project. All had passed the equivalent of a first class Navy flight physical, including complete spine x-rays and a determination of mental status. Some had previous G experience, either operational or in the human centrifuge, others did not. They were taking no significant medications at the time of the program, and were encourage to eat normally, to get sufficient rest, and to avoid alcoholic beverages. The testing was carried out over a period of six weeks.

FIGURE 1-Basic Characteristics of Subjects

Subject	Age	HT/cm	WT/kgms	Body type	Prev.Cent. Experience	Total Flight Time--Pilot		Maximum G Tolerance w/o G suit		with G Suit	
J.R.	42	179.07	87.27	mesoendomorph	+++	5,400	7.0*	(8.5)**	(11)***	8.5*	(12)**
J.R.	25	170.82	58.64	mesoendomorph	+++	-----	6.5*	(9.5)**	(9.5)***	8.5*	(11)**
E.L.	22	170.18	61.82	mesoendoectomorph	+	-----	7.0*	(9.5)**	(10.5)***	8.0*	(11.5)**
L.A.	30	170.18	62.27	mesoendomorph	+++	-----	6.5*	(8.5)**	(12.5)***	8.0*	(12)**
R.M.	22	180.34	78.64	mesoendomorph	0	-----	6.0*	(9.0)**	(9)***	7.0*	(9.5)**
M.M.	34	177.80	68.64	mesoectomorph	++	1,900	8.0*	(10.0)**	(13.0)***	9.5*	(12.0)**
T.M.	20	175.26	69.55	mesoendomorph	+++	-----	5.5*	(8.5)**	--	6.0*	(10.5)**
T.W.	44	167.64	70.91	mesoectomorph	++	-----	7.5*	(12.0)**	(14.0)***	9.5*	(13)**
J.T.	25	175.26	77.27	mesomorph	0	-----	6.0*	(7.5)**	(9.5)***	8.0*	(9.5)**
J.M.	35	170.18	63.64	endomesomorph	++	-----	5.5*	(8.0)**	--	7.0*	(11.5)**

\+ = 1-2 previous projects
++ = 3-4 previous projects
+++ = 5 or more previous projects
* = Tolerance in G_z positions considered
** = Overall tolerance - all positions
*** = H.A.P.P. position

The tests were carried out on the analog computer controlled, double-gimbaled, Dynamic Flight Simulator at the Naval Air Development Center, Warminster, Pennsylvania, which consists of a human centrifuge having a fifty-foot radius arm with the capability of attaining 40 G's in 7 s (seconds).

The subjects were dressed only in leotards, wearing heavy socks and/or tennis shoes. Before and after each session, a complete ECG, urinalysis, CBC with differential, blood chemistries (creatinine, total bilirubin, phosphorus, alkaline phosphatase, uric acid, cholesterol, total protein, globulin, albumin, calcium, BUN, glucose, LDH (cardiac isoenzyme #5 fraction), SGOT, SGPT, CPK, CO_2, and chloride), as well as a complete briefing-debriefing, were done. A standard group of questions was directed to each subject after each run of each session (Figure 2).

FIGURE 2 - Trans Run Questions

How do you feel?
Are you uncomfortable at all?
Do you have any pressure points?
Did you have any difficulty breathing?
Did you notice any peripheral light loss? How much?
Are you becoming more tired or fatigued?
Are you perspiring?
Are you warmer than before?
Are your eyes watering?
Do you have any other comments?

During the runs, EKG (lead II), respiratory rate and rectal temperature were measured, while T.V. monitoring of the face and eyes of the subjects, which were clearly visible under all test conditions, and constant voice communication were maintained.

The conditions of the sessions, as well as G levels attained, are given in Figure 3, and are as follows: seatback angles of 13°, 30°, 45° 60°, and 75° from the vertical, with upper legs at 59° and lower legs at 115° from the vertical, seatback angle at 45° and 75° with the upper legs at 59° and the lower legs at 180° from the vertical, and the H.A.P.P. position-seatback angle of 75° with knees on chest.

FIGURE 3 - Seat configurations for each session. Mean G tolerances for each session.

Run #	1	2	3	4	5	6	7	8
								All angles measured
Seatback Angle	45°	30°	13°	45°	60°	75°	75°	75° from the vertical.
Upper Leg Angle	59°	59°	59°	59°	59°	59°	59°	(legs on chest-
Lower Leg Angle	115°	115°	115°	180°	115°	115°	180°	fetal position)
Mean G Values Attained								
Without G Suit	6.3	5.9	5.6	6.5	7.4	8.2	8.6	11.1
With G Suit	7.8	7.0	6.9	7.9	8.6	10.5	10.7	--

The rate of onset of all runs was 3.5 G/s. The duration of the G plateau (G level) was 15s, with a rate of decay of 2.5 G/s. The G levels were increased in increments of 0.5 to 1.0 G for each successive run, depending upon the state of P.L.L. (Peripheral Light Loss). P.L.L. was measured by means of a 76.2 cm horizontal bar supporting three lights, the two peripheral lights at the ends of the bar being green and the central light red, the green light intersecting an angle with the eye of 54°. The green (peripheral) lights blinked on and off in a pseudo-random sequence with a frequency of from 0.1 to 1.0 s. Each subject was instructed to look at the red light and to press a button each time the green lights were observed, "turning off" these lights in that manner. If the light signal were not answered in a period of two seconds, the centrifuge was programmed to automatically stop. Each subject was exposed to a G level at which P.L.L. occurred, and this was considered to be his "G-tolerance" limit. The session ended at that point. The subjects performed the M-1 maneuver during all runs. After initial P.L.L., a G suit was added, in each session, to find additional G-tolerance limits.

RESULTS:

The changes in G tolerance between the different seatback angles was significant in nearly all cases. There was no significant difference between sessions #1 and #4, so they may be considered together.

FIGURE 3 Session #	Without G Suit	With G Suit
2 vs (1&4)	p = .025	p = .010
3 vs (1&4)	p = .010	p = .005
3 vs 2	p = .025	p = NS
(1&4) vs 2	p = .025	p = .010
(1&4) vs 3	p = .010	p = .005
1 vs 5	p = .005	p = .050
1 vs 6	p = .005	p = .005
2 vs 3	p = .025	p = .005
2 vs 5	p = .005	p = .005
2 vs 6	p = .005	p = .005
3 vs 5	p = .005	p = .005
3 vs 6	p = .005	p = .005
3 vs 6	p = .050	p = .005
4 vs 7	p = .005	p = .005

General Observations Pre-Trans-Post Run:

The mean amount of sleep before each session was 6.8 hours (hrs). The quality was rated as restful (87%), fitful (10%), and poor (3%), the subjects feeling rested (79%), somewhat rested (18%), and not rested (3%), when questioned. G-tolerance was not significantly affected by adverse sleep habits in this series. The mean time since last food intake before each session was 7.5 hr, varying from 1 to 14 hr, depending upon whether the subject had just finished a meal before the session, or had fasted since the evening meal the night before. The average time since alcohol intake, within 24 hr of the session, was 12 hr, ranging from 8-16 hr. Only 38% of the sessions were preceded by such intake. Present physical condition was reported as excellent (37%), good (43%), and average (20%, the frequency of physical exercise being every day (31%), twice a week (28%), and once a week or less (41%). These factors did not significantly affect changes in G-tolerance found in this series.

Pre-session comments reflected a willingness to participate in the centrifuge project, while post-run comments centered primarily on immediate transient dizzyness on egressing the gondola, tiredness, and feelings of well-being (Figure 4). The majority of these symptoms were absent in 24 hr, and all were absent in 48 hr.

Physical examination post-session revealed only petechiae in 70% of the cases (Figure 4), these usually being observed in the lower portions of the trunk (Figure 5).

The subjects estimated their time of "recovery" (to be ready for the next session) from 0 to 48 hr, distributed normally, the mean being 26 hr.

Trans-run comments centered on feelings of tiredness, perspiration, vertigo, difficulty in breathing, warmness, headache, dizzyness, lacrimation, and a variety of pressure point locations (chest, buttocks, and lower back being the most frequent sites). Coughing and vertical nystagmus were also frequently observed by the medical monitor. The relation of these with session number, and the presence or absence of a G-suit, can be seen in Figure 6.

FIGURE 4 - Post-Session Symptomatology and Signs

Symptoms--Immediate Post-Run

Session #	1	2	3	4	Total	% Of Total
dizzy	9	7	7	9	32	31
tired	5	3	2	7	17	17
good, fine, O.K.	3	7	4	0	14	14
relaxed	0	0	1	6	7	7
thirsty	2	1	1	2	6	6
hungry	3	1	1	0	5	5
lower back tenderness	0	0	4	0	4	4
headache	0	1	1	1	3	3
weak	0	0	0	3	3	3
elated	1	1	0	0	2	2
neck pain/stiffness	0	0	1	1	2	2
abdominal discomfort	1	0	1	0	2	2
joint tenderness	0	0	2	0	2	2
"shaky"	0	0	0	1	1	1
difficulty concentrating	1	0	0	0	1	1
general stiffness	0	0	1	0	1	1
					102	

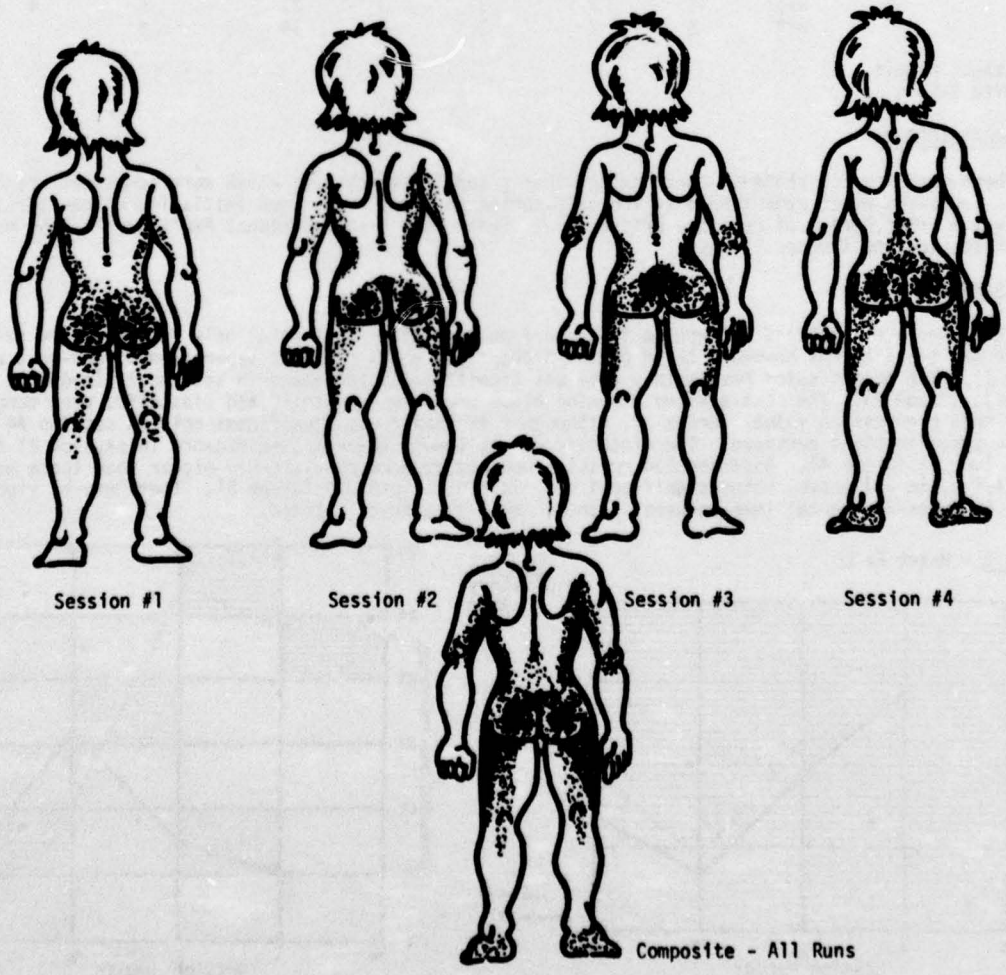
Signs--Immediate Post-Run

petechiae	50%	70%	80%	78%		mean = 69.5%

Symptoms--24 Hours Post-Run

fatigue	0	1	1	0	2
neck pain/stiffness	0	0	2	0	2
difficulty concentrating	1	0	0	0	1
headache	0	0	1	0	1

FIGURE 5 - Petechiae Distribution Post-Session



Session #1 Session #2 Session #3 Session #4

Composite - All Runs

FIGURE 6 - Trans-Run Comments

Session #		1	2	3	4	Total	% Of Sub-Total	% Of Total
perspiration	w/o*	21	14	9	14	58	18	16
	w**	33	18	5	16	72	15	
tired	w/o*	6	10	6	5	27	9	14
	w**	25	19	19	21	84	18	
pressure on chest	w/o*	10	16	6	20	52	16	14
	w**	19	8	9	24	60	13	
warm	w/o*	8	14	14	7	35	11	11
	w**	15	15	11	12	54	11	
pressure points	w/o*	4	9	5	10	28	9	10
	w**	9	16	18	9	54	11	
difficulty breathing (2° to G)	w/o*	3	4	3	10	20	6	6
	w**	6	3	6	11	26	5	
cough	w/o*	5	5	5	2	17	5	6
	w**	7	7	6	10	30	6	
vertigo	w/o*	10	5	3	2	20	6	5
	w**	8	6	4	2	20	4	
lacrimation	w/o*	6	3	1	3	13	4	5
	w**	4	3	8	14	29	6	
headache	w/o*	5	8	4	5	22	7	5
	w**	2	2	2	11	17	4	
dizzy	w/o*	1	5	0	4	10	4	2
	w**	3	3	3	1	9	2	
other	w/o*	12	3	3	3	21	4	4
	w**	5	2	3	4	14	4	

* = Without G-Suit
** = With G-Suit

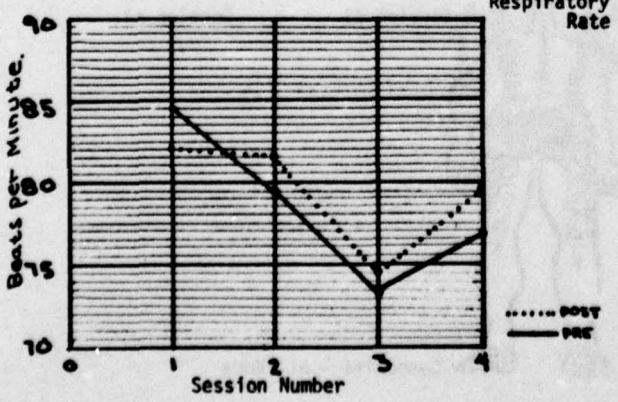
ELECTROCARDIOGRAM:

There were few ECG changes, these being minor p and T wave changes which were completely reversed at 24 hr. Trans-run heart rate tended to increase during the runs, but, upon initiation of deceleration, there was a brief period of relative bradycardia. There were also occasional PVC's-usually not more than one per session and frequently less.

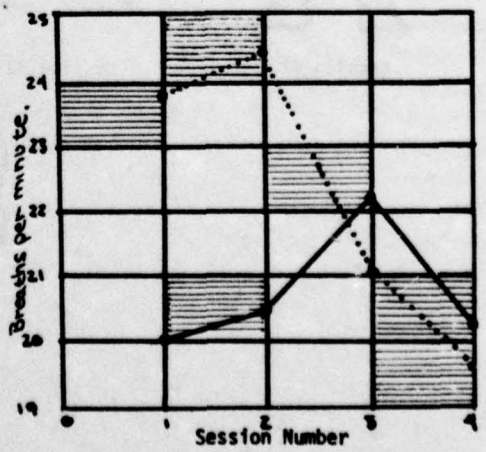
VITAL SIGNS:

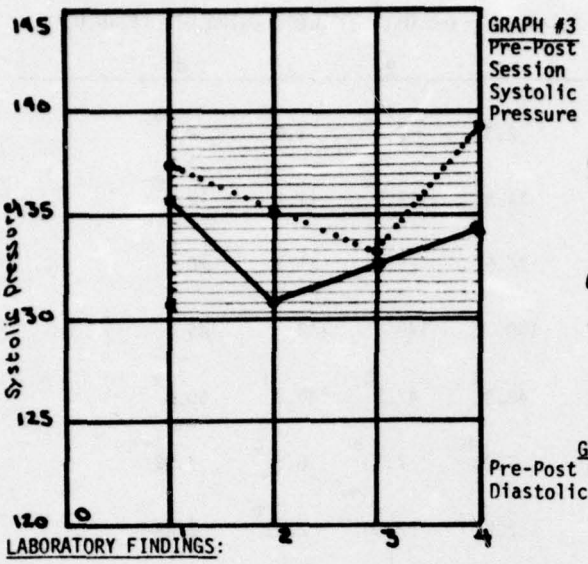
There was a significant difference in the pre-post-session heart rates only in session #4 (p=0.01), but one may see a steady downward trend of the heart rates and a constant separation of pre-post run values (Graph 1). The post-session respiratory rate was significantly increased in session #1 (p=0.05), and #2 (p=0.02), (Graph 2). The post-session standing blood pressures, systolic and diastolic, were consistently higher than pre-session values (Graph 3), although this change was significant only in session #4 (p=0.02), for the systolic blood pressure. The diastolic values tended towards significance in session #1 (p=0.2) and #3 (p=0.1) (Graph 4). Post-session rectal temperatures were consistently higher than those measured pre-session, in all cases, being significant only in run #1 (p=0.05) (Graph 5). There was no significant change in trans-run rectal temperatures, although this same trend existed.

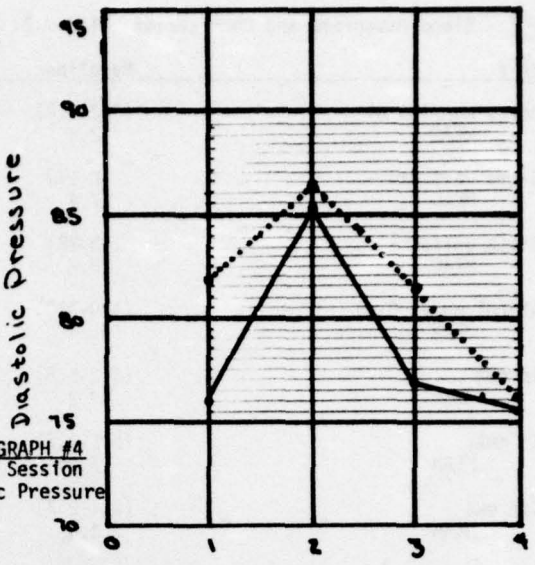
GRAPH 1 - Heart Rate



GRAPH 2
Respiratory Rate



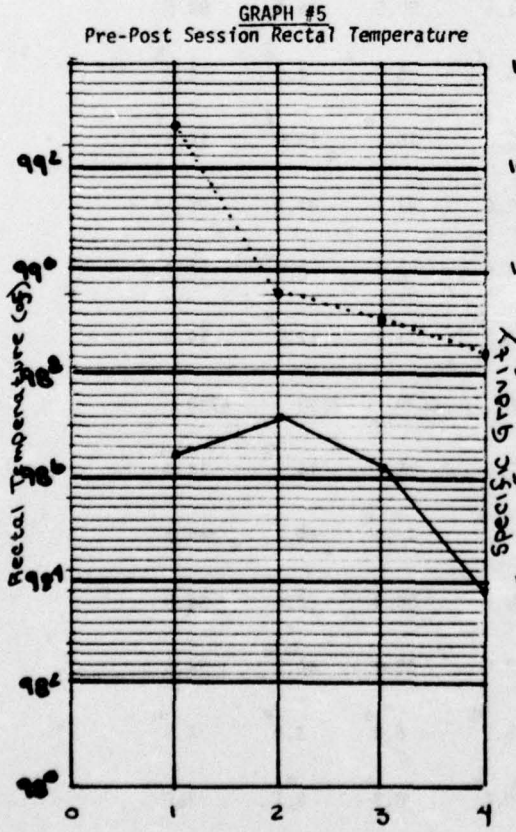




LABORATORY FINDINGS:

There was no significant change in urine specific gravity, although there was a trend towards a more dilute (Graph 6) and a more alkaline urine (session #1 ($p=0.05$), #3 ($p=0.1$)).

The blood data (Figure 7) demonstrated a significant rise in albumin, chloride ions, creatinine, calcium, and BUN over the period of time of the sessions; a significant decrease in values for phosphorus, SGPT, protein, glucose, uric acid, CO_2, hematocrit, and monocytes. Either no significance, or contradicting data were obtained for values of SGOT, cholesterol, globulin, alkaline phosphatase, bilirubin, CPK, WBC, hemoglobulin, neutrophils, lymphocytes, eosinophils, basophils, and immature white cells. The values for the LDH cardiac-isoenzymes obtained during the sessions all fall well within the normal zone.



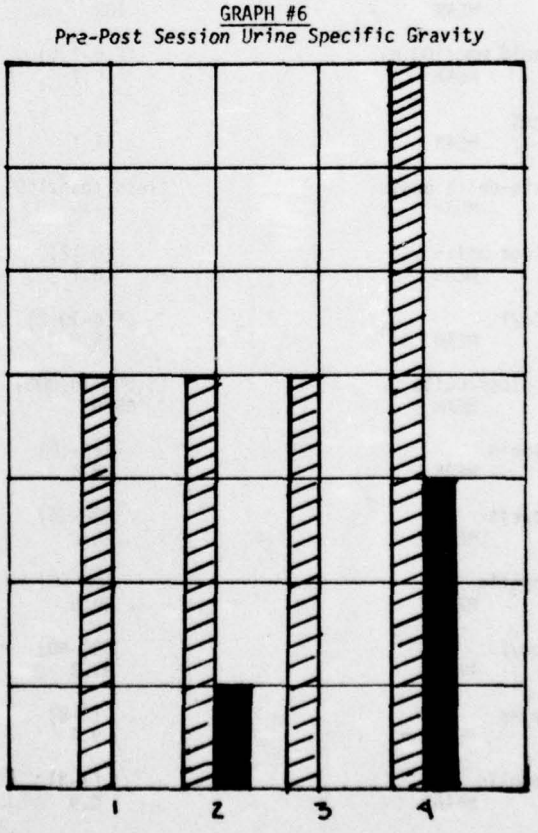


FIGURE 7 - Blood Hemograms and Chemistries *(a)=0.2; (b)=0.1; (c)=0.05; (d)=0.02; (e)=0.01; (f)=0.001

Session #	Baseline	1	2	3	4
Phosphorus mgm/100 ml	(2.3-3.8)				
MEAN	2.8	2.3 c	2.3 c	1.8 f	2.5 b
SGPT Sigma units/ml	(0-21)				
MEAN	27.4	14.5 e	12.3 e	11.7 f	12.8 e
SGOT Sigma units/ml	(0-28)				
MEAN	28.2	24.5	25.7	21.3	20.7
Cholesterol mgm/100 ml	(120-310)				
MEAN	156	150	148	148	149
Albumin gm%	(3.2-5.6)				
MEAN	43.2	48.3 d	47.2 b	49.3 d	50.5 e
Protein gm%	(6.5-7.7)				
MEAN	7.75	7.08 b	7.05 e	6.55 c	7.92
Globulin gm%	(2.1-2.7)				
MEAN	3.4	2.75	2.90	2.03 e	2.82
Chloride mEq/l	(98-109)				
MEAN	87.2	82.3	75.7 b	125.3 b	103.7 e
Creatinine mgm/dl	(0.2-0.6)				
MEAN	0.64	0.84 b	0.89 c	0.87 b	0.96 e
Calcium mgm/dl	(8.6-10.2)				
MEAN	10.6	13.6 f	13.4 f	12.46 d	10.65
Alkaline phosphatase Sigma units/ml	(0.8-3.0)				
MEAN	2.2	2.24	2.23	2.36	2.52
Glucose mgm%	(60-100)				
MEAN	102	101.5	90.5 b	84.0 b	89.5 b
Uric acid mgm/100 ml	(2.6-7.5)				
MEAN	5.7	5.1 c	5.3	4.9 c	4.6 b
BUN mgm%					
MEAN	11.1	15.3 f	15.1 e	15.0 f	14.5 f
LDH beta-delta units	(less than 140)				
MEAN	----	89.6	97.1	91.3	97.77
CPK Sigma units	(0-12)				
MEAN	6.1	7.9	8.1	8.8 b	2.7 f
CO_2 mEq/l	(5.6-10.2)				
MEAN	18.5	16.2 b	11.2 f	12.9 e	19.1
White Blood Cells	(5,000-10,000)				
MEAN	6570	6570	6920	7730	6730
Hemoglobin	(14-18)				
MEAN	15.4	15.4	15.2	16.3 b	14.8 c
Hematocrit	(38-54)				
MEAN	45.2	42.7 c	42.6 c	44.2	44.8
Neutrophils	(40-60)				
MEAN	55.5	56.7	56.0	52.4	54.0
Lymphocytes	(20-40)				
MEAN	31.3	38.0	32.3	40.5 e	34.3
Monocytes	(4-8)				
MEAN	9.4	5.9 d	6.8 e	3.8 e	4.0 e
Eosinophils	(1-3)				
MEAN	0.9	0.2 e	0.9 b	2.1	2.0
Basophils	(4-8)				
MEAN	0.6	0.2	0.0 e	0.2	0.3
Bands	(0-5)				
MEAN	1.3	1.3	0.0 d	1.0	5.1 e

DISCUSSION:

The general conditions, such as amount and quality of sleep, time since last food intake, amount and time of last alcohol intake, present physical condition (subjective), and frequency of physical exercise each week, did not seem to affect the G-tolerance in this series. However, this is a very small series of just ten subjects, with constantly changing conditions. It is felt that a controlled study over a longer period of time, with constant conditions would, indeed, demonstrate changes in the majority of these variables.

Before each session, each subject demonstrated an eagerness to participate. This was felt to be related to the fact that each session was under different conditions, thus affording a new experience to the subjects at each exposure, so that they had a natural curosity to experience the new conditions.

Immediately upon exiting the gondola, each subject reported transient dizzyness, in almost all cases. This was probably due to assuming an upright posture after having been stressed while semi-recumbent. The subjects also reported fatigue, a state which was only reversed by sleeping through the night.[3] After a centrifuge experience, the subjects tended to go to sleep earlier and sleep more soundly and longer than usual. Upon awakening the next morning, they usually feel refreshed, although, as one can see from Figure 3, in some instances the fatigue lasted longer than 24 hr. The post-run feelings of well-being reported by the subjects probably reflected their elation at having successfully overcome a stressful test. There were few physical complaints post-run, with these concerned primarily with back and neck tenderness. Since the G vector in all instances was some component of Gz, the simple exposure to this force was probably enough to stress previously unused musculature. Thus, Figure 3 shows that the majority of such complaints occurred in the 13° seatback angle. The feelings of tiredness and dizzyness occurred more frequently in the 30° and 45° seatback angle conditions, probably because the stress was greater in these positions (high G level achieved, Figure 2), and the dizzyness can be attributed to differences in body position, thus affecting the inner ear mechanisms.

Petechiae usually occur at points of stress or pressure; the subjects wore only leotards and the seat was well padded to reduce,as much as possible, the number of such pressure points.

Figure 4 shows composite views of the petechiae found and their location; considering the G vector, they are located as would be expected.

After each run of each session, the subjects were questioned in a standardized fashion in order to obtain as recently experienced sensations as possible. The answers to such questions depended on the G level experienced, the seat configuration, the time into the session, and whether or not the subject was wearing a G-suit. Greater fatigue, perspiration, and warmth were reported at the higher G levels (higher stress levels) and, consequently, with a G-suit on. Pressure on the chest and difficulty in breathing again were directly related to G level, presence of G suit, and seatback angle. When the G vector was directed more towards the chest, the sensation of pressure and, in some cases, of pain,occurred in the chest region. Pressure points, as such, seemed to be more prevalent with the 13° seatback angle, again at the higher G levels. These were many and diverse, with no special predominance of location. Coughing was seen almost exclusively among the heavy smokers, and may be physiologically explained by the presence of secretions in the tracheal-bronchial tree, induced by the irritative properties of tobacco smoke, and to the inhibition of the normal cleansing mechanism of the tracheal-bronchial tree caused by reorientation of the gravitational field. Vertigo and dizzyness were relatively common, and seemed to follow no particular pattern, although they were probably related to how much the subject moved his head during the exposure. Lacrimation was probably caused by the G vector pressure on the lacrimal glands, but, of course, this cannot be substantiated. Headaches were usually frontal and/or peri-orbital, usually occurring only during each G exposure and disappearing after same. It is difficult to explain this, unless one might postulate that it was caused by increased pressure on the sinuses which were inadequately "depressurizing" under the G force.

For the majority of the sessions, the post-session heart rates were lower than the pre- session heart rates. This may have had a physiological basis, or may simply have been a manifestation of pre- session anxiety. The post- session systolic and diastolic blood pressures demonstrated the expected physiological response to stress of an increase over the pre-run values. The pre-post-session respiratory rates showed no real trend in this series, although the post-session rate "tended" to decrease with number of sessions. The lack of significance of change of rectal temperatures trans-run are reflected in the pre-post run values, although there clearly is a trend that post-run temperatures were higher than the pre-run temperatures. This is borne out by the subjects' repeated reports that they felt progressively warmer throughout the sessions, although the gondola temperature did not change.

As mentioned previously,[4] the post-session urine samples were consistently more dilute and more alkaline, although not significantly so in most cases, probably because of the great variability of individual values. A physiological change in the kidneys at the level of glomerular filtration could have occurred or reabsorption could have increased, thereby resulting in a greater retention of H^+ ions. A variety of other mechanisms could also have been active, in that the concentration of Cl^- ions rose and the CO_2 levels in the blood fell.

The significant increase in albumin and the corresponding significant decrease in total protein, without a significant change in globulin, seems equivocal. Values for globulin did decrease, but not significantly so, probably due to individual variability. Perhaps the increased albumin was caused by a "G" effect on the liver, but the decrease in total protein and globulin is indeed interesting. The increase in creatinine and BUN, and the corresponding decrease in uric acid could indicate a functional renal change, or possibly a muscular reaction. The increase in calcium may indicate some form of skeletal involvement, or functional changes of the parathyroid gland, or perhaps again a renal manifestation. The decrease in values of glucose, contrary to our previous findings[4] may be stress related,--the glucose being utilized a higher rate. The SGPT probably decreased due to the relative acidosis, and we can also see a trend for the SGOT to decrease, although the changes are not significant (table 7). The rest of the blood chemistries demonstrate either equivocal evidence, or no significant change.

As one can see by the above data, there still remains much work to be done to delineate the cause of many of the manifestations noted, physically and biochemically. One may consider this effort but one phase of a beginning, to stimulate more efforts, and more complete efforts, in the future.

The data in Figure 3 demonstrates a definite significant increase in G tolerance with each increase in seatback angle. The position of the lower legs, whether they be vertical or at 115°, however, makes no significant difference in G tolerance. This is important because the PALE (Pelvis and Legs Elevated) position, as originally designed, would require a large cockpit and could cause difficulty at ejection. With the lower legs vertical, the seatback angle being constant, the majority of these problems could be more easily resolved. The G tolerances demonstrated here, at 3.5G/s onset rate, conform to previously reported data at 1 G/s.[5,6]

In order to evaluate the G-protective effects of body positioning, as described in this report, tolerance to increasingly higher levels of sustained accelerations has been studied. While this technique is useful in measuring differences in G-protection afforded by various means under controlled laboratory conditions, it is not representative of the situation experienced by pilots of modern high-performance fighter aircraft. During typical missions of these aircraft, which may last an hour, moderate and low G fields lasting for relatively long periods are broken by short bursts of rapidly changing G levels, the peaks of which may exceed 6G. Because of the brevity of these exposures to very high G levels, the pilot may experience no noticeable dimming of vision; however, the overall effect of repeated G exposures encountered during the mission may result in extreme fatigue, with concomitant decrease in pilot performance.

In an effort to study this problem, we are presently simulating the fighter mission profile just described, while introducing, singly, and in combination, such typical additional stresses as buffet, vibration, noise, temperature changes, etc. Through subjective and objective evaluations of physiological and performance changes, we hope to achieve a better understanding of the roles played by cockpit configuration, body position, and personal protective and survival gear as they are related to producing or alleviating pilot fatigue.

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DISCUSSION

UNTERHARNSCHEIDT (United States): Did you examine the fundus of the eyes in your subjects after a run?

VOGE: Yes. No abnormalities were observed.

MACKENZIE (United States): How great were calcium and creatine changes in the serum?

VOGE: All were within normal limits.

HAMLIN (United States): Could you describe, better, the P-wave changes? Did you infer that size of centrifuge and not G forces is important?

VOGE: No P-wave changes of significance were observed. To the second question, yes. We seldom see premature beats observed by others.

MONESI (Italy): Did you employ any rating scale in order to assess the mental condition of the subject?

VOGE: No, the answers to the questions are a simple "yes" or "no". If a subject replies "yes", he is asked if the symptom is worse than the previous run. Only changes are considered - status quo is not.

CLARKE (United States): Are the observed biochemical changes of clinical significance?

VOGE: The biochemical changes are significant in the paired t test, but all values remained within normal limits. However, for each particular case, (e.g. subject), I consider that these changes are significant.

BALLO (United States): Did the creatinine values come back to normal?

VOGE: Yes, within about 2 - 3 weeks in a non-stressed environment.

MACKENZIE (United States): If there is muscle damage in man as occurs in the pig, this could possibly be a source of the increased creatinine.

CENTRIFUGE ASSESSMENT OF A RECLINING SEAT

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SUMMARY: A reclining seat has been built which would give a pilot a significant increase in acceleration tolerance whilst maintaining adequate forward vision. The effect of anti-G suit inflation has been investigated using three different pressure regimens, and positive pressure breathing (PPB) has been used to counter the added inspiratory effort which resulted from the considerable $+G_x$ acceleration vector. The reclining seat alone gave an increase in tolerance of 1.4G when compared with a conventional seat; anti-G suit inflation afforded a further 1.0 to 1.6G; and PPB a further 1.0G. The combination led to relaxed greyout thresholds which averaged 7.4G (range 6.0 to 8.6G) in 9 subjects. PPB produced a significant increase in vital capacity and restored the expiratory reserve volume to near normal levels. Subjectively, breathing became much easier. The closing volume of the lung was increased by acceleration, but was not significantly affected by PPB. However, the increase in expiratory reserve volume with PPB should lead to less airway closure during tidal breathing, with a consequent increase in arterial oxygen levels and a decreased susceptibility to acceleration atelectasis. It is considered that a seat in which a near supine position is adopted with respect to the G vector, when used in conjunction with an anti-G suit and positive pressure breathing, will result in a G tolerance which is in more accord with the performance of modern military aircraft.

INTRODUCTION

In a previous paper from this Institute (Crossley and Glaister, 1970), the relationship between supination and G tolerance was reported. It was shown that a linear relationship existed between the reciprocal of heart to eye distance and the relaxed peripheral greyout threshold. It was also shown that an anti-G suit was still effective in raising G tolerance even at extremes of supination, and that when protected by both supination and an anti-G suit, a subject's tolerance could be limited by respiratory distress rather than by a visual end point.

As the heart to eye distance decreases as the cosine of the back angle from the vertical, it is not until relatively large angles are used that useful reductions in heart to eye distance are seen. Thus, for high-G protection, a large degree of supination is necessary. This is at variance with the requirement to maintain useful forward vision. A compromise seat was therefore designed incorporating the following features.

a) The back support datum was 60° from the vertical.

b) The buttock and thigh support datum was horizontal.

c) The head was supported at or very near the vertical.

d) The lower legs were inclined downwards at 30° from the horizontal.

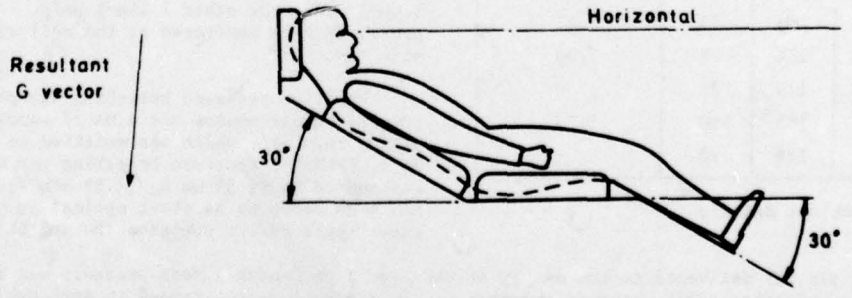

Fig. 1 The reclining seat

From figure 1 the general configuration of the seat can be seen. When mounting it in the centrifuge gondola, the increase in angle of attack of the aircraft during high-G manoeuvres was taken into consideration. This angle was taken as 5° and the seat was actually mounted with the back angled 65° from the vertical, as indicated by the resultant G vector.

The seat was moulded in rigid fibre-glass in two parts, the buttock and thigh support and the back and shoulder support. Shapes were designed for subjects of average to large size and, though the only adjustment available was the distance separating the two parts (from zero to 100 mm) a large range of subjects has been accommodated in the seat in reasonable comfort.

The advantage that a reclining seat of this type has in minimising hydrostatic pressure differences is not restricted to those which occur above the heart, those below the heart are also of lesser magnitude than in a conventional seat. A typical figure for the heart to ankle distance in a conventional seat is 90 cm, whereas the corresponding figure in the reclining seat is 30 cm. This smaller distance would indicate that a lower anti-G suit pressure gradient could be used. The lowest gradient available to us on a commercial valve was 0.8 psig (40 mm Hg, 5.5 kNm^{-2}) per G, the actual pressure at any level of acceleration being 0.8n-1 psig, where n = total acceleration in G. Since an anti-G suit has effects other than simple counterbalancing of hydrostatic pressure gradients (increasing peripheral vascular resistance, for example), two higher anti-G suit pressure gradients were also investigated. These were 1.0n-1 psig and 1.25n-1 psig (6.9n-6.9 kNm^{-2} and 8.6n-6.9 kNm^{-2}).

During $+G_z$ acceleration, the changes in vital capacity (VC) and expiratory reserve volume (ERV) are small, VC reducing at $+6G_z$ to about 90% of its $+1G_z$ value. During $+G_x$ acceleration, the increased weight of the anterior chest wall is such that at 6G the VC falls to approximately 50% of its 1G value, and the ERV becomes virtually zero above $+3G_x$ (Glaister, 1970). This progressive reduction in vital capacity is the limiting factor in the G protection which may be afforded by supination, regardless of the amount of cardiovascular support which may be given by an anti-G suit or other measure. Whilst the seat used in this evaluation does not completely supinate the subject, it does impose a large $+G_x$ vector, so some respiratory difficulties would be expected.

Positive pressure breathing (PPB) has been used experimentally to aid acceleration tolerance. In a conventional position, PPB elevates the systemic blood pressure with smaller fluctuations than do repeated M1 manoeuvres. (Burton et al, 1974). The use of PPB in a reclining seat offers a twofold advantage. It should raise the systemic blood pressure, as in any other body orientation, and it should aid the lifting of the anterior chest wall and so increase the VC and ERV. An additional factor might be that, by increasing ERV, PPB would improve the ventilation of the dependent lung zones and so minimise the development of shunting and absorptional atelectasis.

In order to evaluate these factors, relaxed peripheral greyout thresholds were determined in the conventional seated position, and in the reclining seat using various anti-G suit pressure gradients and PPB. Changes in VC, ERV, tidal volume (TV) and closing volume (CV) were also investigated.

METHODS

Eleven healthy male subjects were used. Only three had significant pilot experience, but all were experienced centrifuge subjects. Appropriate details are given in table 1.

Subject	Age	Height (cm)	Weight (kg)	Vital Capacity 1G reclined (l)
DG	41	183	77	4.84
BL	32	184	85	4.62
RW	26	182	77	5.72
DR	41	182	87	4.48
MG	28	182	78	4.60
FP	45	184	78	5.20
SC	23	183	73	5.42
PG	34	184	78	5.00
AB	34	175	76	-
DM	22	185	90	5.15
BB	26	188	70	-

Table 1. Subject details

Greyout threshold was defined as the level of acceleration, which when maintained constant for 15 seconds, caused the subject to lose peripheral vision for at least 5 seconds whilst maintaining central vision. Test lights were mounted at eye level and the angle subtended by the peripheral lights was 45°. Acceleration was applied with an onset rate of 1.0G sec^{-1}. Subjects were instructed to relax. Threshold measurements were carried out on 9 subjects.

When appropriate, an RAF Mk 6C anti-G suit was inflated from a compressed air bottle using Hymatic anti-G valves. One delivered either 0.8n-1 psig or 1.0n-1 psig, the other 1.25n-1 psig. Inflation pressures were monitored at the delivery hose/suit hose junction.

Positive pressure breathing was provided from a compressed air source via a Mk 17 panel mounted oxygen regulator which was modified to deliver 5 mm Hg (0.6 kNm^{-2}) pressure breathing per G up to a maximum of about 35 mm Hg (4.67 nKm^{-2}). These levels had been shown to be about optimal in preliminary experiments on two subjects (DG and BL).

Breathing air was delivered to the man by an RAF Type P or Q mask. Mask pressure was monitored from a tapping using a Statham P23Gb pressure transducer. As the P or Q mask tended to leak during the 6G PPB runs, a rubber mouthpiece was used for lung volume measurements. Volumes were obtained by continuous integration of flow derived from a Fleisch pneumotachograph mounted immediately downstream of the mouthpiece.

Lung volume measurements were made in 7 subjects at 1, 4 and 6G and at 4 and 6G with PPB. At 4 and 6G subjects wore an anti-G suit inflated at 1.25 n-1 psig. Subjects were requested to breathe quietly for a few breaths, then to exhale to RV, inspire fully to total lung capacity (TLC) and then exhale again to RV. They then breathed quietly until the end of the runs.

Lung closing volumes were measured by the argon bolus technique. A 50 ml bolus of argon was introduced from a syringe into the breathing system close to the lips when the subject had exhaled to RV. The subject inspired slowly to TLC and then exhaled slowly to RV again. During this latter exhalation the expired argon concentration was monitored by a remote mass spectrometer (Micromass Q 701). Due to the 28.5 m long sample line, argon concentrations were delayed some 10 secs compared to the flow information. This delay was measured during each manoeuvre and subsequently, argon concentration against volume plots were made using a digital delay line to synchronise the two parameters. Closing volumes were estimated

from the argon volume plots by two independent observers, the point taken being a sudden increase in argon concentration from the plateau value (Fig. 6).

RESULTS

Threshold Determinations

The individual threshold estimations are shown in Table 2, and the average findings are shown graphically in figure 2. Increasing anti-G suit pressure led to a significant increase in threshold both with and without PPB ($p < 0.001$). PPB produced a highly significant increase in threshold ($p < 0.001$).

Subject	Conventional seat	Reclining seat							
		No PPB				PPB			
		anti-G suit pressure							
	0.0	0.0	0.8n-1	1.0n-1	1.25n-1	0.0	0.8n-1	1.0n-1	1.25n-1
DG	3.8	5.4	6.7	6.8	7.8	6.5	8.4	8.4	8.6
BL	3.4	4.4	5.6	6.0	6.0	5.2	7.2	7.2	7.2
RW	3.4	4.2	6.2	6.8	6.8	5.0	7.4	7.2	8.2
MG	3.0	4.6	5.0	5.3	5.7	4.8	5.8	6.1	6.0
DR	3.6	5.0	5.6	5.8	6.0	5.4	6.6	7.0	7.1
BB	3.4	4.4	5.2	5.9	6.0	4.8	5.6	6.4	6.4
AB	3.4	5.8	6.6	7.2	7.2	6.4	7.6	8.0	8.4
FP	3.8	5.4	6.4	7.2	6.6	5.8	7.5	7.7	8.0
SC	3.6	4.8	6.0	6.0	6.7	5.4	6.4	6.6	7.0
Mean	3.49	4.89	5.92	6.33	6.53	5.48	6.94	7.18	7.43
SD	0.25	0.55	0.61	0.68	0.68	0.64	0.91	0.76	0.91

Table 2. Relaxed greyout thresholds for 9 subjects under various conditions of posture, positive pressure breathing (PPB) and anti-G suit pressurisation.

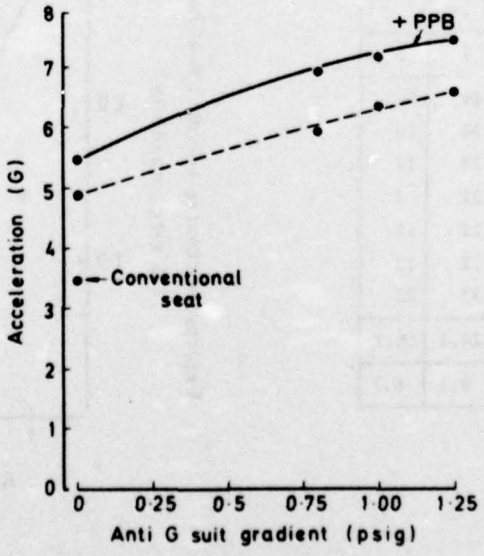

Fig. 2 Mean relaxed greyout thresholds

Vital Capacity

Individual results expressed as percentages of average vital capacity reclining at 1G (5 estimations for each subject) are shown in Table 3. Average findings are shown graphically in figure 3. Acceleration led to a fall in VC ($p < 0.01$), but the decrease was less with PPB ($p < 0.01$).

Subject	No PPB		+ PPB	
	Acceleration level			
	4	6	4	6
DG	37	24	72	45
BL	42	30	65	43
RW	42	23	56	37
DR	53	36	60	27
MG	49	35	58	48
FP	62	29	62	54
SC	70	46	85	55
Mean	50.7	31.9	65.4	44.1
SD	11.8	7.9	10.1	9.8

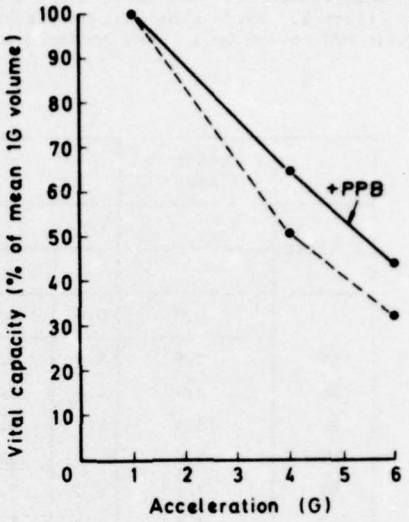

Table 3 and Figure 3. Vital capacity, expressed as a percentage of the 1G reclining value, at 4G and 6G with and without positive pressure breathing (PPB).

Expiratory Reserve Volume

Individual results expressed as percentages of average vital capacity are shown in Table 4, and the average results are shown graphically in the corresponding figure. Without PPB, acceleration led to a significant fall in ERV ($p<0.05$), but PPB increased ERV towards the 1G levels ($p<0.001$).

Subject	No PPB			+PPB	
	Acceleration level				
	1	4	6	4	6
DG	23	0	0	29	20
BL	29	4	0	28	19
RW	35	10	0	28	17
DR	25	0	0	22	2
MG	26	9	9	13	14
FP	22	6	0	12	19
SC	31	18	18	37	22
Mean	27.3	6.7	3.8	24.1	16.1
SD	4.6	6.3		9.1	6.7

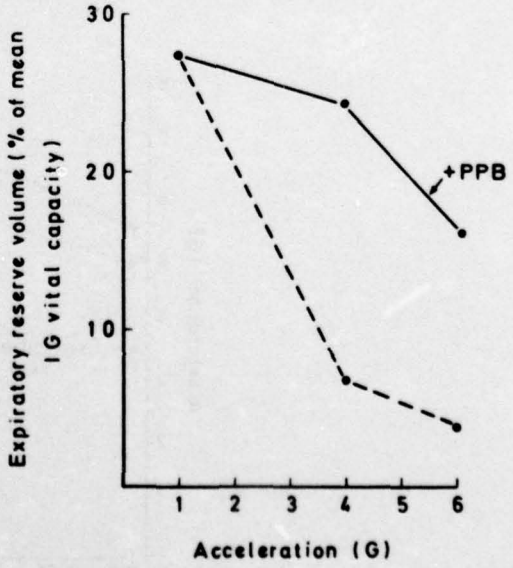

Table 4 and Figure 4. Expiratory reserve volume, expressed as a percentage of the 1G reclining vital capacity, at 1G, 4G and 6G with and without positive pressure breathing (PPB).

Tidal Volume

Individual results expressed as percentages of average vital capacity are shown in Table 5. Average findings are shown graphically in figure 5. Tidal volume was decreased by acceleration ($p < 0.05$), but PPB had a variable effect (not statistically significant).

Subject	No PPB			+PPB	
	Acceleration level				
	1	4	6	4	6
DG	16	5	4	15	12
BL	17	12	10	15	10
RW	13	15	10	9	9
DR	16	13	11	11	11
MG	17	18	11	14	11
FP	20	15	19	17	19
SC	13	15	11	13	15
Mean	16	13.2	10.9	13.4	12.4
SD	2.4	4.1	4.4	2.7	2.46

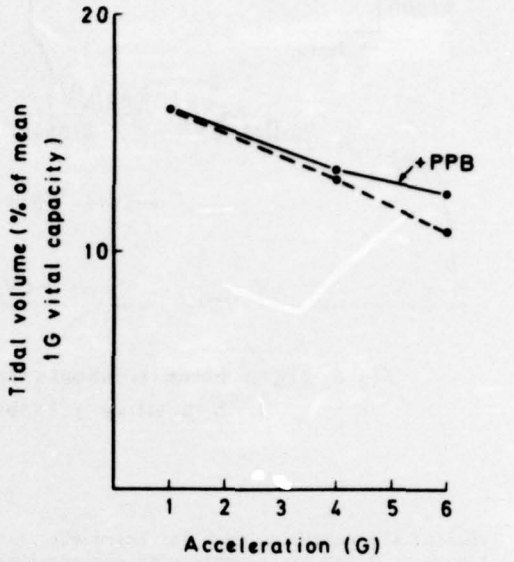

Table 5 and Figure 5. Tidal volume, expressed as a percentage of the 1G reclining vital capacity, at 1G, 4G and 6G with and without positive pressure breathing (PPB).

Closing Volume

Closing volumes for the various experimental conditions are given in table 6, expressed as percentages of the subjects' 1G reclining VC. CV was increased threefold at 4G ($p<0.001$), but was unaffected by PPB. At 6G, CV could not be measured, since it was greater than the available VC, but as this was greater than the closing volume at 4G, a further increase must have occurred. Thus, the table quotes minimum values. Again, PPB had no demonstrable effect.

Subject	1G	4G	6G	4G PPB	6G PPB
RW	12.1	37.2	>36.6	27.0	>30.7
PG	33.3	>46.8	>40.8	35.6	>48.0
FP	4.0	20.4	>33.7	>48.0	>44.4
DG	13.2	35.3	>44.4	53.6	>57.1
DM	3.8	54.7	>66.3	50.5	-
MG	12.8	45.9	>38.8	59.6	>41.5
BL	21.3	47.8	>43.5	46.3	>56.1
Mean	14.4	41.2	>VC at 6G	45.8	>VC at 6G
SD	10.3	11.3		11.1	

Table 6. Lung closing volumes, expressed as a percentage of the 1G reclining VC, at 1G, 4G and 6G with and without positive pressure breathing.

The inspiratory distribution of the argon bolus is also influenced by acceleration, it being the existence of dependent airway closure which ensures that the bolus preferentially labels alveoli in the superior lung. To eliminate this inspiratory effect, runs were carried out on two subjects with the bolus inspired to TLC at 1G. The centrifuge was then run up to 4G and the argon washout recorded. The effect of pressure breathing (25 mm Hg) was also investigated at 1G in these subjects and the results are given in table 7. Again, it is clear that 4G has greatly increased CV and that PPB has had no demonstrable effect. Typical traces are illustrated in figure 6 to show the relationship between CV and other lung volumes for that subject. Thus, at 1G (left hand panel, figure 6), closure is seen to occur some 0.7 l above RV, but since the ERV was greater than this (1.1 l), airways would have remained open during tidal breathing. At 4G with PPB (right hand panel), closing volume increased to 2.3 l, but ERV was little changed at 1.3 l. Closure would have persisted throughout tidal breathing. However, in the absence at PPB, ERV was zero in this subject at 4G and more of the lung would have been subject to closure during tidal breathing.

Subject	Conditions during bolus washout			
	1G	1G PPB	4G	4G PPB
DG	17.8	19.8	54.9	52.0
BL	14.9	14.0	50.0	55.5

Table 7. Closing volumes, expressed as a percentage of the 1G reclining VC, with argon boluses inspired at 1G.

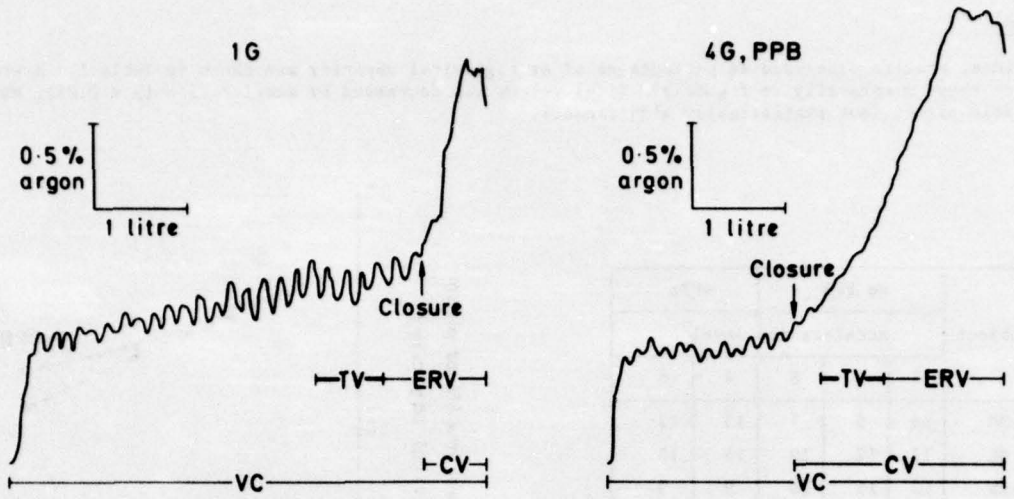

Fig.6 Argon bolus washouts recorded at 1G (left panel), and at 4G with positive pressure breathing (right panel)

DISCUSSION

Use of the reclining seat increased the mean greyout threshold value by 1.4G from 3.49G to 4.89G. The figure of 3.49G agrees well with our previous experience for unprotected man in a conventional seat. The figure of 4.89G, when compared with earlier data (Crossley and Glaister, 1971), suggests that the composite angles built into the seat equate with a single back angle of about 65° from the G vector.

Again, in conformity with earlier findings, the use of an anti-G suit raised the G thresholds by amounts similar to those seen with a conventional seat, that is, some 1 to 1.6G. Even the lowest pressure gradient used was greater than that estimated to counteract vascular hydrostatic pressure increases. Thus, vascular transmural pressures in those parts of the body covered by the anti-G suit would have been less during acceleration than at 1G. Since the higher gradients gave added G-protection (Fig. 2), an increase in peripheral vascular resistance must have been an important factor.

In the absence of pressure breathing, most subjects reported difficulty in breathing and chest pain at the higher G levels. Several subjects even held their breath for the duration of the runs, a procedure hardly conducive to the measurement of relaxed greyout thresholds.

It was noted in runs with anti-G suit and PPB that the visual symptoms of greyout tended to take longer to develop than the 5s from peak G normally experienced in a conventional seat. Thus, in the reclining seat and using both protective measures, aircrew would have more warning of impending blackout and would have adequate time to tense their muscles.

Two subjects commented adversely, one because of inflation of the middle ear - a complication of PPB per se rather than of the reclining seat; the other because of vascular engorgement of his arms. Subsequently this subject was found to have developed petechial haemorrhages. If such a problem were commonly encountered it might be necessary to offer counter pressure to the arms, perhaps by an extension of the anti-G suit.

The intrapulmonary pressure needed to overcome the weight of a 30 mm thick anterior chest wall would be of the order of 2 mm Hg (0.3 kNm^{-2}) per G, but it has been shown, during $+G_x$ exposures, that intrapulmonary pressure at any given lung volume is increased by 4-5 mm Hg (0.5-0.7 kNm^{-2}) per G (Glaister, 1970). This is similar to the value found to be preferred in preliminary experiments in which both lower and higher pressures gave chest discomfort and difficulty in breathing at the higher G levels.

On its own (no anti-G suit), PPB resulted in an increase in relaxed greyout threshold which averaged 0.6G (table 2), despite the fact that it would have increased the pooling of blood into the lower limbs. This effect would be countered by an anti-G suit, and the combination of suit plus PPB led to several runs in excess of 8G. Subjectively, PPB made breathing much easier. Thus the active expiratory effort required during pressure breathing at 1G was provided passively during acceleration by the increased weight of the chest wall, and each inspiration started from the lung's relaxation volume. Furthermore, at 4G, this volume was similar to that observed at 1G (table 4) so that the mechanics of breathing would have been very close to normal and the work of breathing would have been similar to that of normal breathing at 1G.

Closing volume results from a gradient of transpulmonary pressure which causes a gradient of decreasing alveolar size down the lung such that dependent alveoli attain their regional RV at a lung volume greater than overall RV. Since the transpulmonary pressure gradient is most easily accounted for on the basis of lung weight, CV is increased by acceleration. This has been observed in $+G_z$ exposures (Jones, Clarke and Glaister, 1969) as well as in $\pm G_x$ exposure (Glaister, Ironmonger and Lisher, 1975).

The present results indicate a threefold increase in CV at 4G (to an average of 2.1 l in absolute terms) together with a decrease in ERV (table 3). Thus, considerable right-to-left shunting would be expected together with absorptional atelectasis if oxygen were breathed. Note that breathing oxygen cannot correct arterial hypoxia caused by a right-to-left shunt. The situation is worse at 6G and, since CV is then greater than VC, subjects could not prevent shunting or atelectasis by voluntarily breathing at an increased lung volume. PPB has no effect on CV (tables 6 and 7) but, by increasing the ERV, could greatly reduce the degree of shunt and atelectasis at 4G. The average relationship between CV and ERV + TV is given in table 8, from which the likely magnitude of right-to-left shunting and sensitivity to absorptional atelectasis may be predicted.

	Experimental conditions				
	1G	4G	6G	4G PPB	6G PPB
VC	100	61.2	43.4	70.0	49.0
CV	14.4	41.2	>43.4	45.8	>46.3
ERV + TV	43.3	19.9	14.7	37.5	28.5

Table 8. Average relationship between CV and ERV plus TV, expressed as a percentage of the reclining VC at 1G.

CONCLUSIONS

Two practical limitations to the reclining seat indicate that alone it cannot be used to raise G tolerance by a large amount. These are the need to maintain adequate forward vision which effectively limits the feasible reduction in heart to eye distance to one half of the conventionally seated distance, and the respiratory distress which becomes a limiting factor above approximately 6G.

The use of an anti-G suit provides adequate support to the systemic cardiovascular system up to a point where breathing is severely embarrassed. In order to maintain respiration, positive pressure breathing has been used and found to be effective up to levels of acceleration where a cardiovascular end point i.e. peripheral greyout, is obtained. Its use also maintains an expiratory reserve volume under acceleration which will mean that although right-to-left shunting due to an increase in closing volume will occur in the lung, the magnitude of the shunt and resultant decrease in arterial oxygen saturation will be of lesser degree.

Further studies are indicated to investigate whether higher levels of pressure breathing may be tolerated in this position, whether some form of arm counter pressure is required and to examine the subjective acceptability of the seat and pressure breathing to aircrew. Direct measurement of arterial oxygen tension and of sensitivity to acceleration atelectasis are also required to substantiate the predictions.

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DISCUSSION

UNTERHARNSCHEIDT (United States): Did you record the EEG or are you aware of such recordings in the literature?

LISHER: No.

HAMLIN (United States): Why was there no drift in phase III of the closing curve (CV)?

LISHER: This may be due to the fact that both the inspiratory and expriatory phase of respiration took place at 4G. When inspiration takes place at 1G and expiration takes place at the G level in which we are concerned, more classical traces are obtained.

VARENE (France): My question concerns the use of PPB during accelerations: According to the authors, is the improvement of the tolerance to G stress observed in those experiments related to respiratory effects (improvement of respiratory exchange) or to circulatory effects (increase in blood pressure) of PPB?

LISHER: The increase in G tolerance is almost definitely attributable to the effect on the systemic blood pressure. The respiratory effects are, however, a very beneficial secondary effect.

EWING (United States): If the effect of the reclining seat is to change the acceleration vector on the chest wall from $+G_z$ to $+G_x$, and if the effect on the lungs is due to chest wall compression of lungs due to the $+G_x$ acceleration, could this be alleviated by using some means to prevent lateral expansion of the chest, thus preventing chest wall deflection and resulting in decreased lung compression? And, could coupling of PPB with the above result in a greater acceleration protection than either one individually?

LISHER: First, it should. Second, there are no data but it should.
(Editor's note - Earlier work on space couch design (NADC) showed the value of the splinting effect of lateral support in preventing chest pain. The practicality of providing such protection in a conventional cockpit has not been demonstrated.)

VOGE (United States): We have reported, as have others, that the limiting factor to G tolerance on many occasions is pain in the chest. Does positive pressure breathing lessen the chest pain?

LISHER: From personal experiences I would say that it definitely does alleviate the chest pain associated with high $+G_x$ acceleration. During our evaluation other subject's comments have confirmed this view.

CORONARY FLOW AND MYOCARDIAL BIOCHEMICAL RESPONSES TO HIGH SUSTAINED $+G_z$ ACCELERATION

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SUMMARY

In order to determine directly the myocardial response to $+G_z$ acceleration, miniature swine were used as the experimental subjects. Some of the animals underwent surgical implantation of flow probes around the left circumflex coronary artery and a solid-state pressure transducer in the left ventricular cavity. All of the unanesthetized instrumented subjects were exposed to multiple $+G_z$ acceleration levels for 60-120 seconds (3, 5, 7, 9, 11 $+G_z$) on the USAF School of Aerospace Medicine human centrifuge. Other subjects were exposed to a single acceleration level (9 $+G_z$) for 120 seconds and the hearts removed for biochemical analysis 1-2 hours later. Mitochondria and a lysosomal fraction were isolated from the left ventricle of all animals. Mitochondrial analysis of ADP:O ratio, respiratory control index (RCI), oxygen uptake (QO_2) and calcium uptake were made. Free and bound acid phosphatase measurements were made in the lysosomal fraction. Left circumflex coronary artery flow (LCCF), heart rate (HR), left ventricular pressure (LVP), and the rate of rise of LVP ($\dot{P}$) were measured in the instrumental animals. LVP and HR increased at all levels of acceleration studied while $\dot{P}$ increased initially but would decline later. LCCF decreased at all levels of acceleration stress. The mitochondrial ADP:O ratio and the RCI were unchanged but the QO_2 and calcium uptake were increased at 9 $+G_z$. Free acid phosphatase increased at the same level of acceleration.

INTRODUCTION

The increased capability of high performance aircraft has necessitated a new investigation into the cardiovascular response to high sustained $+G_z$ acceleration levels. In man during $+G_z$ acceleration exposure, abnormalities in the electrocardiogram and some arrhythmias have been noted (1, 2, 3, 4). The changes in the electrocardiogram have been associated with the S-T segment and are felt to suggest myocardial ischemia. For many reasons other measurements in man have not been made at high $+G_z$ levels, thus the use of an animal model is appropriate. Miniature swine have been used in this regard (5, 6) and found to show similar changes to that observed in man. The coronary vasculature in the swine is much more analogous to man than most other animals. High levels of $+G_z$ acceleration in swine have been found to be associated with subendocardial hemorrhage and pathological changes in the myocardial cell (7). Evidence indicates that, in both man and swine, myocardial ischemia must be considered as a consequence of high sustained $+G_z$ acceleration.

Myocardial ischemia is the result of a dramatic reduction or cessation of coronary flow to all or discrete portions of the myocardium (8). When coronary flow becomes the limiting factor in the delivery of oxygen to the myocardial cells, the contractile mechanism begins to fail following apparent changes in the cell membrane that allow the inward leakage of sodium ions and other cations and the outward leakage of protein molecules. At a certain step in the process of cell leakage, the enzymes that are contained in lysosomes are released and begin to destroy other proteins through their hydrolytic actions. The energy producing organelles are also affected by this process. Mitochondria increase their respiratory activity in response to the reduction in oxygen in an attempt to increase the amount of energy available for cellular processes. Thus, the key mechanisms in cellular dysfunction and arrhythmia production (9, 10) with high sustained $+G_z$ acceleration may be a reduction in coronary flow. In the conscious miniature swine, it should be possible to detect changes in the coronary flow with $+G_z$ acceleration and at the same time determine if myocardial ischemia may be occurring either through a reduction in total coronary flow or a divergence of flow away from the endocardium (8). These results could be correlated with biochemical changes associated with ischemia such as lysosomal and mitochondrial function.

MATERIALS & METHODS

The present study was made up of two groups of miniature swine. Group 1 animals were used to determine the effects of acceleration on left ventricular pressure and coronary flow, while Group 2 animals were used to study the relationship of acceleration to the changes in lysosomal and mitochondrial function.

Group 1

This group of animals was anesthetized with sodium pentothal and surgical anesthesia was maintained with a mixture of oxygen, nitrous oxide, and halothane. The heart was exposed through the left 5th intercostal space. The left circumflex coronary artery was exposed for a length of 3 cm along the atrioventricular groove. An electromagnetic flow probe was placed around the vessel, as was a balloon occluder distal to the flow probe. A solid-state pressure transducer was positioned in the left ventricle through a stab incision in the apex of the heart. A silastic catheter was placed in the left atrium via the left atrial appendage. The wires from the two transducers and the two silastic catheters were passed out of the chest through the 6th intercostal space and left in a subcutaneous pouch. The chest incision was carefully closed to prevent adhesions between the lungs and chest wall. The animals were allowed to recover for 30 days before being used for any experimental procedure. At the end of this period, the lead wires were exposed under local anesthesia and taped to the animals' backs.

The conscious animals were placed in a fiberglass couch and positioned on the animal arm of the USAF School of Aerospace Medicine centrifuge. The animals were minimally restrained during the experimental

period. The wires from the two transducers were connected to appropriate electronics. The electrocardiogram was measured from limb leads or from the pressure transducer. The case of the miniature solid-state pressure transducer plus an additional ground lead can be used for this purpose. Left circumflex coronary artery flow (LCCF), left ventricular pressure (LVP), heart rate (HR), the rate of rise of left ventricular pressure ($\dot{P}$), mean left circumflex coronary flow (MLCCF), and the level of acceleration ($+G_z$) were recorded on both a direct-writing oscillograph and magnetic tape. The animals were exposed randomly to levels of 3, 5, 7, and 9 $+G_z$ acceleration with a rapid onset rate of 1 G/sec. Peak levels of acceleration were maintained for either 120 seconds at the lower levels or 60 seconds at the higher levels. The animals were allowed a minimum of 20 minutes for recovery between runs. Before each acceleration profile, the LCCA was briefly occluded to establish zero flow. The resulting hyperemic response was allowed to disappear before beginning the profile. At the termination of the experiment, the wires were taped to the sides of the animals for future use.

The left ventricular pressure transducer and the electromagnetic flow transducer were calibrated prior to implantation. The zero reference for both transducers was established at the beginning and at the termination of each experiment. The sensitivity of both transducers has not been found to vary over the course of the time involved with these experiments.

Group 2

The animals in this group were exposed to a single $+G_z$ profile. The animals were placed in the couch and loosely strapped in place. Limb leads were connected for the measurement of the electrocardiogram. The animals were exposed to 9 $+G_z$ for 120 seconds following a rapid onset rate of 1 G/sec. One to two hours following this acceleration profile, the animals were anesthetized with sodium pentothal and the hearts rapidly excised. Samples were taken from the left ventricular free wall for biochemical analyses.

Mitochondria were isolated from the samples of left ventricular free wall and their respiratory activity and oxidative phosphorylation capabilities were measured polargraphically (11). Respiratory substrate-supported mitochondrial calcium uptake was measured by dual-beam spectroscopy. Acid phosphatase was utilized as a heart lysosomal marker enzyme. Left ventricular free wall tissue homogenates were prepared in 0.25M sucrose using a blade homogenizer. Enzyme activity was partitioned into sequestered (lysosomal) and free (soluble) fractions using modifications of a differential centrifugation procedure and assay (12). The ratio of acid phosphatase specific activity present in these fractions (soluble/lysosomal) provided an estimation of lysosomal membrane integrity. The results of this portion of the study were compared to results obtained from unoperated-control and operated-control animals.

RESULTS

Physiological Responses

The miniature swine seemed to tolerate the exposure to the various levels of acceleration used in this study. The peak levels of acceleration were randomized for each animal so as to minimize the effect of the first exposure level on the subsequent results. At 9 $+G_z$, all of the animals appeared to remain conscious. The criterion for this was the kicking and grunting behavior of the animal. Closed circuit television allowed the observer to watch and hear the animal during the various profiles. Two types of responses were observed in this study. The first can be seen in Figure 1. In this animal, the heart rate increased and remained elevated during the entire profile. In the second response, seen in Figure 2, heart rate increased with acceleration but then very abruptly decreased into a bradycardia. At most of the high acceleration levels (+7 and +9), some degree of bradycardia was noted. The severity of this bradycardia varied greatly between animals. All of the measured parameters were allowed to return to control values prior to any succeeding runs.

After the animal had been placed on the centrifuge and before each level of acceleration, control values were taken for heart rate, left ventricular systolic and diastolic pressure, left circumflex coronary flow, and the maximum rate of rise of left ventricular pressure. The maximum rate of rise of the left ventricular pressure was used as an index of the contractile state of the myocardium. The average values with one standard error of the mean were found to be: HR, 97 $\pm$ 3 bpm; LV systolic pressure, 156 $\pm$ 8 mm Hg; LV diastolic pressure, 4 $\pm$ 1 mm Hg; LCCF, 58 $\pm$ 5 cc/min; and, $\dot{P}$, 2472 $\pm$ 164 mm Hg/sec.

The results of exposure to 3, 5, 7, and 9 $+G_z$ acceleration for various periods of time can be seen in Table 1. The average heart rate increased with acceleration, but the magnitude of increase became less with successive increases in the level of $+G_z$ acceleration. At the point of measurement, the left ventricular systolic pressure increased, but it must be noticed that this was not a transmural pressure. Coronary flow decreased at all levels of acceleration studied. There did seem to be a tendency for coronary flow to increase during individual acceleration profiles but in most of the studies remained below control values. The contractile index of the left ventricle increased with acceleration. The increase seemed to be less with higher levels of acceleration. At times, there appeared to be waves in the coronary flow that coincided with changes in heart rate.

Biochemical Responses

Biochemical measurements from miniature swine hearts were established in unoperated and operated-control animals. In control heart mitochondria, three parameters were measured: ADP:O ratio, respiratory control index (RCI), and the rate of mitochondrial oxygen uptake during State 3 respiration (QO_2). The ADP:O ratio is a measure of the efficiency of ADP phosphorylation and was found to be 3.2 with glutamate-malate as the substrate and the average RCI was 6.1. The State 3 respiration (QO_2) is the active rate of respiration for phosphorylation and is indicative of the amount of active enzymatic protein present in the inner mitochondrial membrane. In the control heart preparations, a value of 185 natoms/min/mg mitochondria protein was found. These values fall within acceptable normal limits. It is important to note that there were no differences between the unoperated and operated-control animals.

In the animals exposed to 9 $+G_z$, a marked increase in active respiratory rate in the presence of ADP (State 3) was found in the mitochondria. The average value was 285 natoms/min/mg mitochondria protein. The oxidative phosporylation (ADP:O) and RCI were unchanged in these animals.

Calcium transport by the mitochondrial inner membrane is an energy linked process. This measure of mitochondrial function may be another way of assessing the functional integrity of mitochondria. The concentration of calcium necessary to produce the maximum velocity of calcium uptake was 150 μ mol while the actual rate of calcium uptake was approximately 200-250 nmoles/min/mg mitochondrial protein. Instrumentation of the animals was found to have no effect on these parameters. Significant increases in active rates of calcium transport were observed in the mitochondria from animals exposed to 9 $+G_z$ accleration. The average calcium uptake in mitochondria isolated from these hearts was 280 nmoles/min/mg mitochondrial protein.

The lysosomal fraction of the heart was analyzed for the specific activity of alkaline phosphatase and compared to the alkaline phosphatase activity of the soluble fraction. Lysosomal fraction activity averaged 11.2 $\pm$ 0.9 (standard error of the mean) while the soluble fraction averaged 12.1 $\pm$ 1.0 (SEM) nmoles/min/mg protein. Instrumented and uninstrumented animals were not significantly different with respect to lysosomal enzyme activity. The soluble fraction/lysosomal fraction ratio in control animals was 1.08. 9 $+G_z$ acceleration drastically reduced the specific activity of the lysosomal fraction and elevated the activity in the soluble fraction. Lysosomal fraction activity was 9.7 $\pm$ 0.4 (SEM) while the soluble fraction was 32.4 $\pm$ 1.3. These enzyme responses resulted in approximately a 2-fold increase in the soluble fraction/lysosomal fraction specific activity ratio. The loss of enzyme activity from the membrane-bound lysosomal fraction and the increased soluble fraction activity suggests that the integrity of the lysosomal membrane had been disrupted by acceleration. The loss of lysosomal membrane integrity was apparently a generalized phenomenon throughout the left ventricle since nearly identical results were observed in epicardial and endocardial samples.

DISCUSSION

The major area of concern in the current study was the relationship between coronary blood flow, myocardial intracellular function, and acceleration stress. Previous reports (7) indicate the presence of subendocardial hemorrhage in miniature swine subjected to various levels of $+G_z$ acceleration. It also had been pointed out that some type of myocardial necrosis was found in other areas of the myocardium. The question thus arose does the myocardial cell become hypoxic and/or ischemic during exposure to high sustained $+G_z$ acceleration or if the mechanical forces were severe enough to cause the microscopic damage.

Coronary flow studies in unanesthetized and anesthetized dogs (13, 14) have found a decrease in the coronary blood flow with exposure to low levels of $+G_z$ acceleration. In the unanesthetized miniature swine, coronary flow was found to be reduced at all levels of acceleration, as measured in the left circumflex coronary artery. Coronary flow should have increased due to the increase in the contractile state of the myocardium and the increase in heart rate. Both heart rate and contractility are major determinants of myocardial oxygen consumption (15, 16) and would normally contribute to an increase in coronary flow. The real question then becomes the lack of increase in coronary flow during $+G_z$ acceleration. Perfusion pressure of the coronary vessels will influence flow; however, during acceleration, aortic root pressure is likely to be elevated due to 1) the hydrostatic column effect and 2) compensatory mechanisms which maintain head level arterial pressure. The increased heart rate reduces the diastolic period thus tending to reduce coronary flow. In conscious miniature swine, Denn (17) has found a linear increase in coronary flow with increasing heart rate up to 240 bpm. Therefore, heart rate does not seem to contribute to the decrease in coronary flow during $+G_z$ acceleration stress. The tension within the myocardial wall of the left ventricle will cause changes in the coronary flow patterns. With the beginning of isovolumic systole, tension increases and the coronary arterial transmural pressure decreases. The decrease in transmural pressure will cause a decrease in coronary flow during each cardiac systole. During $+G_z$ acceleration, the left ventricular wall tension may be increased as the result of 1) increased aortic root pressure, 2) increased pleural pressure, and 3) deformation of the heart by a caudal movement from the accelerative forces. All of these factors would tend to decrease coronary flow. The caudal movement of the heart toward the diaphragm has been seen by Sandler (personal communication) while studying anesthetized dogs via cineangiography. Since the arch of the aorta is tethered by the branches arising from it, the ascending aorta may be stretched. A possible constriction of the coronary artery ostia may increase the blood inflow resistance during high levels of acceleration. The increased resistance would reduce coronary flow. The coronary vascular bed has an abundance of alpha-adrenergic receptors (18). Alpha-adrenergic receptors will cause vasoconstriction when activated by either circulating catecholamines or the sympathetic nervous system. The neurogenic component of a constrictor mechanism may be activated by heart displacement or by other receptors located in the cardiopulmonary region.

Myocardial oxygen consumption would be expected to increase with increases in heart rate, contractility, and myocardial wall tension. Since coronary flow was found to be reduced below control values at all levels of acceleration studied, the increased demand for oxygen can only be met by an increase in extraction of oxygen from the coronary blood. Myocardial oxygen consumption measurements have not been made to date but usually myocardial oxygen extraction does not change a great deal under a wide variety of conditions (15). Myocardial ischemia and/or hypoxia would seem to be existent under these conditions. This conclusion agrees with the data from some human studies (1) in which changes in the S-T segment of the electrocardiogram have been felt to be synonymous with myocardial ischemia.

Hypoxia and/or ischemia exert marked effects on intracellular systems of cardiac muscle. Lysosomal enzyme activation is elevated in infarcted heart tissue following coronary artery ligation (19). Acute anoxia also increases the proportion of lysosomal enzymes present in the free form within the heart (20). In the current study, a tremendous increase in free lysosomal activity was found at 9 $+G_z$ which suggests some type of myocardial ischemic insult. This would agree with the reduction in coronary flow found during the studies conducted in the instrumented animals. Depressed mitochondrial function would be expected in hearts subjected to hypoxic and/or ischemic insult (21, 22). In the present study, mitochondrial function was elevated which would mitigate against hypoxia and/or ischemia in the acceleration

stressed heart. An increase in intracellular calcium concentration (23) may contribute to the increased mitochondrial activity seen in 9 $+G_z$ stressed animals. This may occur through an increased release of catecholamines in the heart or an increase in the level of circulating catecholamines (24, 25). Other subcellular systems may be affected by brief transient ischemia such as that seen with $+G_z$ acceleration. These systems may contribute to the increased mitochondrial function and cannot be ignored.

In summary, high sustained $+G_z$ acceleration in miniature swine results in a decrease in coronary blood flow and an increase in the average heart rate and contractility while at 9 $+G_z$. An increase in free lysosomal enzymes and an increase in mitochondrial function were found also. These changes may be associated with some type of ischemic damage to the myocardium resulting from the reduction in coronary flow. The reduction in coronary flow may not be the sole factor responsible for the ischemic damage, and other factors such as catecholamines and mechanical forces must be considered. However, a transient ischemic condition may represent the underlying basis for the myocardial cell death reported by Burton (7). Recovery from this insult requires more than 1-2 hours since a portion of the present study was accomplished in this time period. The current study emphasizes the need for more definition of the transient ischemic period under these conditions, and major efforts are being made to accomplish this goal.

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DISCUSSION

SEM-JACOBSEN (Norway): The bradycardia is similar to what I found in pilots who black out. It would be interesting to measure the EEG at this time to see if the animals actually went unconscious.

STONE: We have not done this yet, but it should be done.

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TABLE 1

The average values for heart rate (HR), left ventricular pressure (LVP), the maximum derivative of left ventricular pressure ($\dot{P}$), and the mean left circumflex coronary flow (LCCF) expressed as the percent of the absolute control values in response to $+G_z$ acceleration. The values were taken at the indicated times after reaching peak acceleration levels. The numbers in parentheses are ± one standard error of the mean.

G	T (sec)	H.R.	LVP		LCCF	$\dot{P}$
			Systolic	Diastolic		
3	60	202 (22)	138 (23)	64 (15)	77 (27)	172 (20)
	120	219 (25)	146 (33)	53 (21)	53 (14)	126 (4)
5	30	217 (21)	151 (27)	50 (20)	58 (18)	170 (22)
	60	169 (25)	151 (23)	62 (20)	60 (18)	140 (12)
7	30	148 (22)	173 (22)	127 (9)	80 (22)	144 (13)
	60	167 (29)	152 (20)	101 (14)	54 (9)	112 (16)
9	30	145 (21)	146 (45)	77 (19)	78 (26)	116 (11)
	60	93 (24)	145 (70)	136 (36)	85 (27)	120 (26)

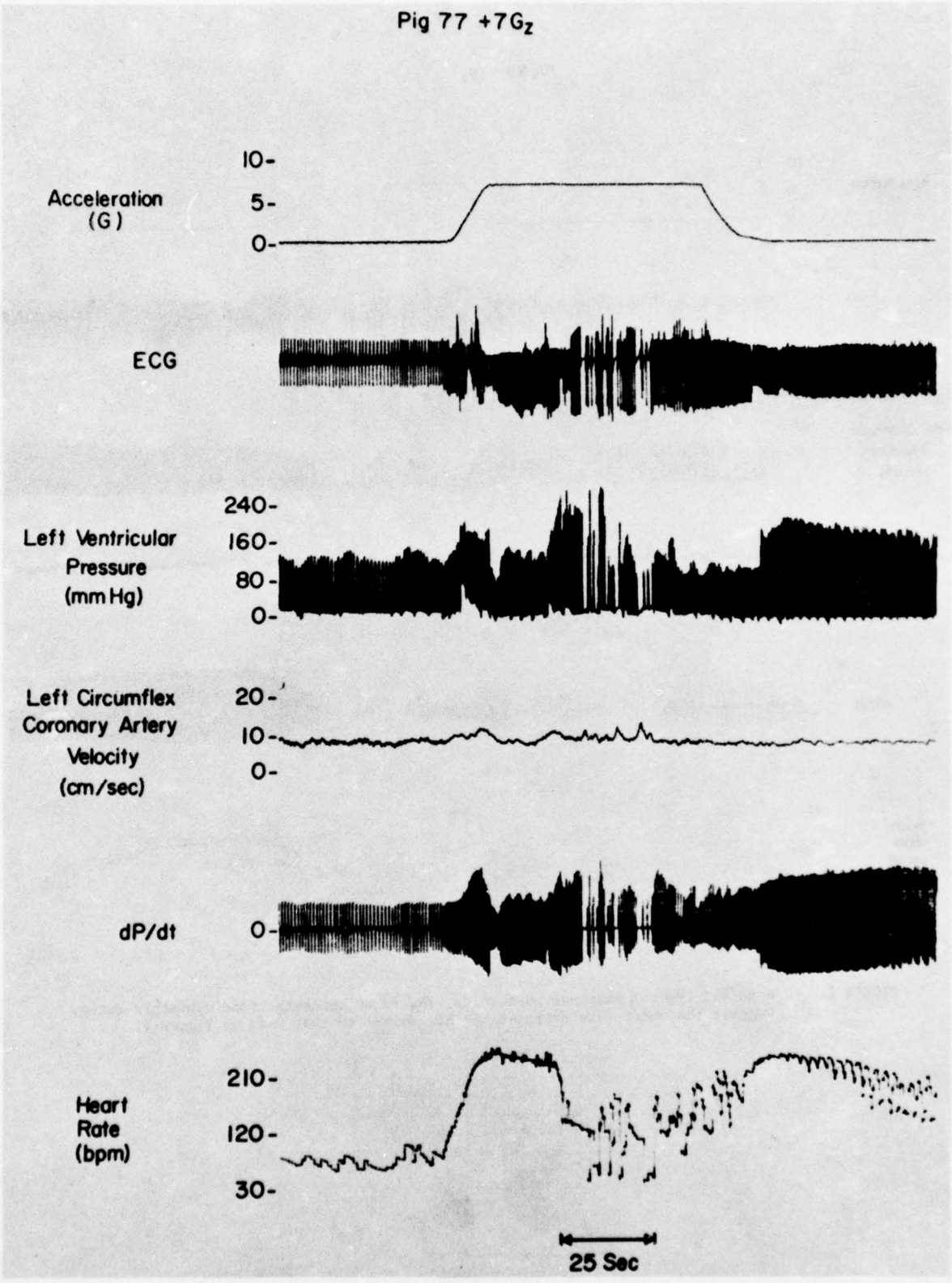

FIGURE 1. A typical response pattern to $+7G_z$ in an unanesthetized miniature swine. Note the heart rate response to the acceleration profile.

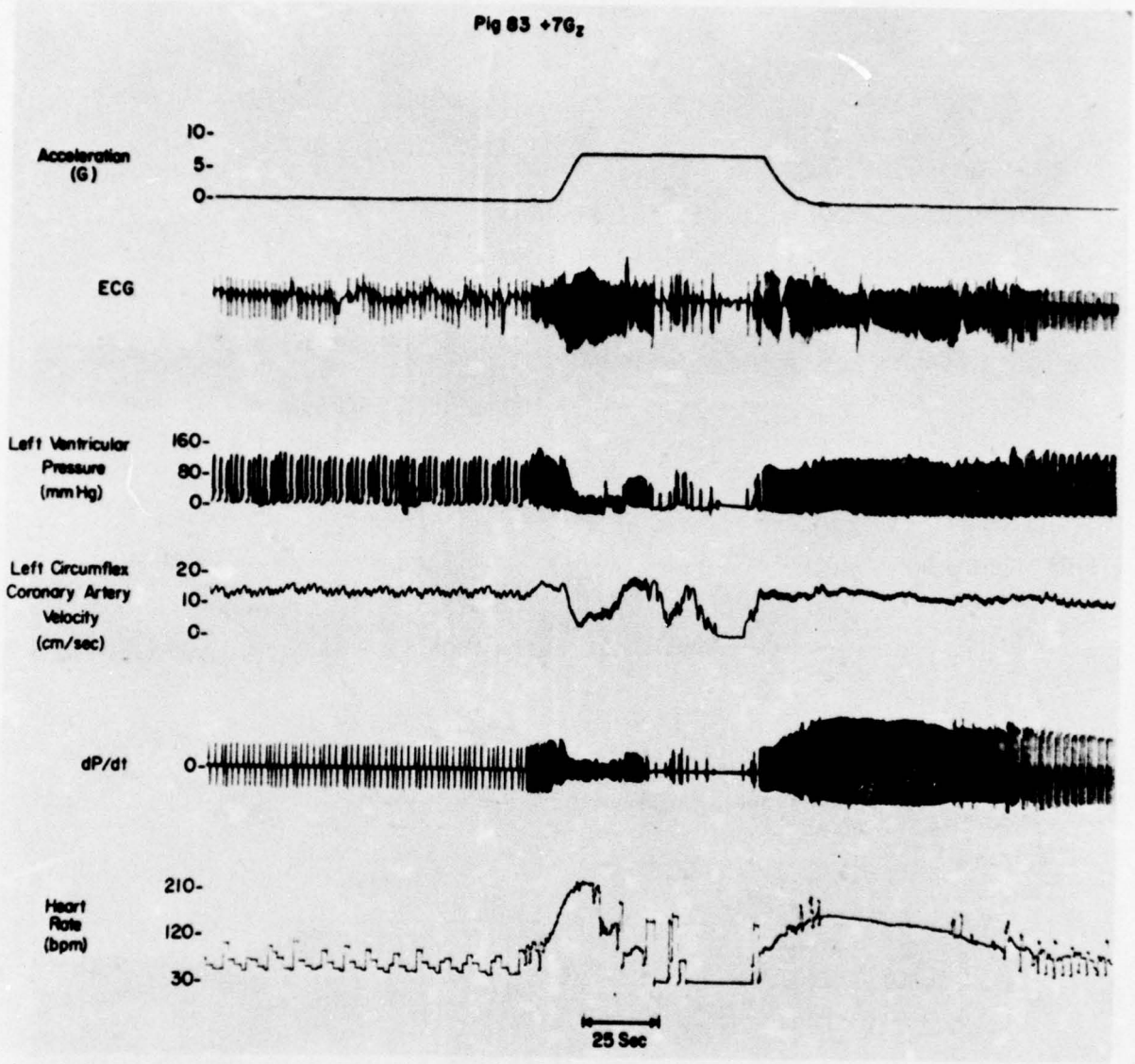

FIGURE 2. A second typical response pattern to $+7G_z$ in an unanesthetized miniature swine. Compare the heart rate response of this animal to that seen in Figure 1.

EFFECT OF SUSTAINED +Gz ACCELERATION ON CARDIAC OUTPUT AND FRACTIONATION OF CARDIAC OUTPUT IN AWAKE MINIATURE SWINE

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Effects of sustained $+G_z$ on cardiac rhythm and output, and on fractionation of cardiac output (CO) were studied in 12 miniature swine centrifuged, while awake, to either $+3G_z$ or $+5G_z$. CO and its subfractions were measured by injecting radiolabeled microspheres into the left atrium. Percentage of CO perfusing most organs fell precipitously during $+5G_z$; while that to the heart increased by twofold and that to the pelvic musculature remained nearly constant. At $+3G_z$ percentage perfusing most organs fell, but that to heart and all skeletal muscle rose twofold. When regional flow decreased, it decreased most to the eye, and next to liver, cerebrum, and renal cortex. It decreased least to the midbrain, spleen, renal medulla and gut. The profound changes in CO and fractionation of CO in awake miniature swine subjected to $+G_z$ may represent a summation of: reflex response, a "waterfall" effect, or deformation of nutrient arteries.

Ventricular arrhythmias in man and swine and subendocardial hemorrhage in swine have been reported following sustained $+G_z$ accelerations.[1] Loss of vision and syncope are common sequel to such acceleration in all species studied.[2] The latter two findings have been attributed to reduced blood flow to organs drained by the force of acceleration. The origin of both the ventricular arrhythmias and subendocardial hemorrhages are obscure; but they may result from alterations in myocardial blood flow. For this reason, the following studies were conducted on miniature swine--a species suggested to serve as a model for the effects of centrifugation of man.[3]

Materials and Methods

This study was conducted on 8 healthy 1-1/2 year female miniature swine. Three days before centrifugation they were anesthetized with sodium thiopenthal, and anesthesia was maintained with 3% halothane in 97% oxygen administered through an endotracheal tube. A silastic catheter was placed with its tip in the descending aorta through the left carotid artery; while the tip of a second silastic catheter was placed in a pulmonary vein via a large transseptal needle inserted through the left jugular vein. Free ends of both catheters were exteriorized at the nape of the neck, and catheters were filled with a solution of heparin. Animals were returned to cages and made uneventful recoveries within 24 hours.

Three days after installation of the catheters, pigs were tied in dorsal recumbency in a plaster cast that was fixed to one arm of a centrifuge. To ascertain that all catheter-tips were as positioned initially, the catheters were filled with radiopaque medium and their positions were verified by fluoroscopy. Electrodes forming ECG leads I, II and III were attached and those leads were monitored continuously on a direct-writing oscillograph.

Cardiac output and fractionation of cardiac output were conducted during a control period and after 30 seconds of centrifugation to either 3 or 5 $+G_z$. With a special totally occlusive pump which rolled solution from a saline reservoir through tubing that contained exactly 1 ml of solution with known activity of carbonized microspheres, 15 microns in diameter and labeled with Ce-141 (for the control period) or Sr-85 (during the period of centrifugation). This pump injected the 1 ml of solution in the tubing and rinsed that volume with 15 ml saline over a 15-second period. At a separate time, we demonstrated that virtually all of the microspheres were rinsed from the tubing by the volume of saline pumped in 15 seconds. Simultaneous with the onset of injection of microspheres into the left atrium, a second roller pump withdrew blood from the descending aorta at a flow rate of precisely 30 ml/min. This served as a reference flow, such that the activity recovered in the reference sample reflected a flow of 30 ml/min. Thus, if we recovered 2 microcurie activity in the reference sample, and 4 microcurie activity in 10 grams of kidney, it indicates that the flow to the kidney was 4/2 times 30 ml/min/10 grams, or 6 ml/gram/min. We measured the amount of activity injected using a dose-calibrator that was calibrated with the deep-well counter for relative efficiencies for the two isotopes used and estimated cardiac output as:

(total activity injected/activity in reference sample) x 30 ml = ml/min

In a separate study, cardiac outputs measured by the reference sample method described above were within 15% of those estimated simultaneously by indicator-dilution methods using indocyanine green dye. No systematic difference between green dye and microspheres techniques existed.

After the control period, pigs were accelerated within 15 seconds to the G-level prescribed, and after mantaining that level for 15 more seconds, the second dose of microspheres was injected. The reference sample was collected for 30 seconds to assure that all of the microspheres had cleared the arterial circulation.

Immediately after the last injection and sampling during centrifugation, the pig was returned to 0 G_z, a lethal amount of sodium pentobarbital was injected intravenously, and many organs or fractions thereof were removed. Weighed aliquots of each organ were counted in a deep-well scintillation detector and the percentage of cardiac output that traversed each aliquot was estimated from the percentage of activity recovered from that aliquot divided by the total amount of activity that was injected. The total blood flow to that aliquot was estimated by multiplying the percentage of cardiac output traversing the aliquot by the total cardiac output. That was expressed, always, as ul/g/min. The heart was divided into basilar,

midbasilar, midapical and apical sections by three cuts parallel to the base. The non-apical sections were divided such that: the right ventricular freewall was cut into endocardial and epicardial segments; the interventricular septum was cut into right, middle, and left segments; the left ventricular freewall was cut into endocardial, middle, and epicardial segments.

Results

Cardiac Output: Cardiac output decreased from a control of 180 (S.E. 24) ml/kg/min to 76 (20) and 49 (14) ml/kg/min during 3G and 5G, respectively. All differences were significant ($p < 0.01$).

Perfusion of Organs Other Than Heart: Perfusion of all organs other than skeletal muscle decreased precipitously and significantly ($p < 0.01$), and decreased, always, more during 5G than for 3G (Table 1). At 3G, neither cerebral nor midbrain blood flow decreased. At both accelerations, blood flow to thigh muscle increased to the same extent while blood flow increased at 3G to intercostal muscle, but decreased precipitously at 5G.

Perfusion of the Heart: By the method used in this investigation, total flow to the heart was not measured and only the perfusion to reasonably small aliquots of myocardium sampled. Table 2 shows mean blood flow to 25 regions of the heart during a control period (for all pigs) and during $3G_z$ and $5G_z$ accelerations for 4 pigs each. Blood flow to all aliquots of heart but to the left atrium at $3G_z$ and basilar sections of the right ventricular epicardium at $3G_z$ increased during $+G_z$ accelerations. Table 3 shows the ratios of perfusion between the various layers (endocardial, middle and epicardial) of both freewalls and of the septum during accelerations. It can be observed that blood flow per gram of septum increases from right ventricular face toward left ventricular face at all levels of acceleration. For the right ventricle, endocardial flow is slightly greater than epicardial flow for all regions. For the left ventricular free-wall, distributions of flows between outer and inner layers depends upon the section (whether apical or basilar) that is examined. For example, for the basilar-third, the middle segment is always greater than the endocardial segment which is always greater than the epicardial segment. For the middle third of the ventricles, segments from endocardium to epicardium are fairly similar during the control period , endocardial and middle portions are perfused similarly, with the epicardial third being perfused with significantly less blood. During $+5G_z$ acceleration, the middle third tended to increase, but this increment was not significant statistically. For the apical-third of the ventricular freewalls, at 0 and $+5G_z$ the endocardium was perfused better than the middle which was perfused better than the epicardium; however, at $+3G_z$ there was no endocardial-epicardial gradient of perfusion of significance.

Discussion

Striking alterations in cardiac output and distribution of cardiac output were documented in this study of the distribution of microspheres injected at varying $+G_z$. Flows measured by this methodology are correct if sufficient numbers of microspheres are injected to recover statistically significant numbers from the subunits of each organ sampled. [4,5] In addition, the microspheres were injected over a 10-second period into the left atrium or pulmonary vein. This permits mixing and averaging over several cardiac cycles. Total cardiac output measured by the reference sample technique is probably correct at higher cardiac outputs and when the reference sample is drawn at a sufficiently high rate (30 ml/min) as in this study. At the lower cardiac outputs, this method is less proven, although the duration of sampling certainly provided for collecting blood during the entire period that microspheres occupied the arteries. That is, sampling began before the microspheres were injected and ended at least 15 seconds after complete entrapment in the capillaries of the systemic circulation. Blood flows to skin, as measured by this method are probably inaccurate because total flow and therefore numbers of microspheres recovered are too small for statistical analysis.

Studies of perfusion to relatively small units of organs would be more reliable using microspheres 8 microns in diameter rather than the 15 micron spheres used in this study.[6] Recent investigators, however, suggest that although 8 micron spheres are distributed more closely to RBC's, 15 micron spheres are distributed close to 8 micron spheres and give a much more reliable flow than 25 micron microspheres.

It is interesting to conjecture about possible reasons for alterations in distribution of cardiac output. First, it appears that flow to heart and brain is preserved at the expense of other organs. This must indicate an autoregulatory mechanism since the brain is an organ which by virtue of its physical position with respect to heart and acceleration should suffer the greatest ischemia. Possibly a high adrenergic release produced vasodilation in these organs; or, as has been shown for the heart, hypoxemia may produce locally active vasoactive substances.

That blood flow to the eye decreases the most and earliest is rather easy to understand, since intraocular pressure is normally between 15 and 20 torr, and a normally lower gradient between arterial and eye-capillary exists. Blood flow to skeletal muscles of the thigh and intercostals increased during moderate acceleration; but that to intercostal then decreased markedly during greatest acceleration. The initial increase to skeletal muscle may result from struggling and increased work performed and tension generated by these muscles (this was shown by artifacts on the ECG), but the fall in perfusion of intercostal muscle and relative preservation of perfusion by thigh muscle may result from hydrostatic effects of acceleration. That is, because of the position of the intercostal muscles and thigh muscles to the heart, $+G_z$ acceleration should favor flow to the thigh and discourage flow to the muscles at heart level or above.

Results similar to that obtained for skeletal muscle were obtained for perfusion to skin from the face as opposed to that from the thigh. At moderate acceleration skin flow to the face fell precipitously; while that to the thigh was maintained--even in the presence of a reduction in cardiac output. Although numbers of microspheres recovered from skin were too low for statistically significant treatment, the trend observed is consistent with the idea that regions away from the acceleration force suffer greater reductions in perfusion than regions towards the force. Thus, facial skin had drastic reduction in flow at any level of $+G_z$; while thigh skin had a reduction only during highest $+G_z$. During moderate acceleration

the reduction in flow caused by the reduction in cardiac output must have been counterbalanced by the increase in perfusion pressure contributed by the added acceleration.

Why in all instances of even the moderate acceleration blood flow to the liver fell so markedly is equivocal. Possibly the nutrient vessels became kinked due to displacement of the organ; or possibly the ability of the vasculature of the liver to autoregulate is less potent than for other organs investigated. The former explanation is less appropriate since the spleen is an organ even more mobile than the liver and it is reasonable that its nutrient vessel would be kinked more than those for the liver.

Alterations in regional perfusions due to accelerations in these swine mimic those produced by exercise alone in which muscular, cardiac, and, to a lesser extent, cerebral flows are maintained or augmented at the expense of abdominal visceral organs and skin.[7,8]

In a subsequent paper the effects of acceleration on cardiac rhythm and rate will be discussed, and the alterations in cardiac output may have been totally a consequence of those determinants. Extreme brady arrhythmias developed during the initial 30 seconds of acceleration, and the fall in cardiac output appeared to parallel the bradycardias.

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Animals involved in this study were procured, maintained and used in accordance with the Animal Welfare Act of 1970 and the "Guide for the Care and Use of Laboratory Animals" prepared by the Institute of Laboratory Animal Resources, National Research Council.

TABLE 1

CARDIAC OUTPUT (ml/kg/min) AND REGIONAL BLOOD FLOW (ul/g/min)
for 0, +3, and +5 G_Z

	Control		+3G_Z		+5G_Z	
	X	SE	X	SE	X	SE
Cardiac Output	180	24	76	20	49	14
Liver	685	135	21	15	20	15
Spleen	1630	388	769	311	129	113
Duodenum	164	14	56	27	32	27
Renal Cortex	3420	454	501	169	183	101
Renal Medulla	717	288	100	33	37	21
Cerebrum	440	38	437	117	82	21
Midbrain	288	37	319	61	102	51
Intercostal Muscle	42	9	125	52	10	10
Thigh Muscle	53	17	142	45	157	87
Facial Skin	8	3	2	1	0	0
Thigh Skin	11	2	4	1	2	1
Eye	593	111	8	2	1	1

TABLE 2

BLOOD FLOW (ul/g/min) for 0, +3, and +5 G_Z

		Control $\bar{X}$	+3G_Z $\bar{X}$	+5 G_Z $\bar{X}$
BASE	RA	200	700	500
	LA	400	500	700
	RVEP	600	700	1250
	RVEN	600	900	1550
	RS	850	1550	2450
	MS	1150	1900	2400
	LS	1200	2400	2300
	LVEN	1000	1700	2200
	LVM	1250	1700	2650
	LVEP	900	1500	2100
MID-BASE	RVEP	550	1200	1350
	RVEN	650	1250	1500
	RS	900	1250	1950
	MS	1200	1650	2200
	LS	1350	2500	2900
	LVEN	1250	2150	3050
	LVM	1250	1750	2700
	LVEP	950	1650	2050
MID-APEX	RV	800	1500	1750
	RS	800	1750	2100
	MS	1250	1700	2450
	LS	1550	2400	2300
	LVEN	1450	1900	3250
	LVM	1200	2000	2800
	LVEP	800	1850	1950
APEX	A	1000	2200	2250

TABLE 3

RATIOS* OF FLOWS TO VARIOUS REGIONS OF VENTRICLES

	Control		+3Gz		+5 Gz	
	X	SE	X	SE	X	SE
REP_1	1.00	0.00	1.00	0.00	1.00	0.00
REN_1	1.14	0.23	1.48	0.28	1.29	0.25
RS_1	1.00	0.00	1.00	0.00	1.00	0.00
MS_1	1.49	0.16	1.20	0.18	1.15	0.14
LS_1	1.62	0.28	1.53	0.35	1.36	0.32
LEN_1	1.00	0.00	1.00	0.00	1.00	0.00
LM_1	1.27	0.07	1.08	0.14	1.23	0.09
LEP_1	0.92	0.08	0.93	0.09	0.98	0.05
REP_2	1.00	0.00	1.00	0.00	1.00	0.00
REN_2	1.15	0.12	1.05	0.22	1.29	0.25
RS_2	1.00	0.00	1.00	0.00	1.00	0.00
MS_2	1.30	0.17	1.45	0.31	1.01	0.15
LS_2	1.63	0.27	2.10	0.36	1.30	0.22
LEN_2	1.00	0.00	1.00	0.00	1.00	0.00
LM_2	0.98	0.12	0.81	0.04	1.11	0.29
LEP_2	0.86	0.09	0.92	0.10	0.88	0.27
RS_3	1.00	0.00	1.00	0.00	1.00	0.00
MS_3	1.66	0.16	1.05	0.11	1.35	0.25
LS_3	1.75	0.19	1.37	0.21	1.40	0.27
LEN_3	1.00	0.00	1.00	0.00	1.00	0.00
LM_3	0.90	0.07	1.09	0.15	0.85	0.12
LEP_3	0.62	0.08	1.02	0.20	0.54	0.11

*Heart is trisected (1, 2 & 3) from base to apex, and gradients of flows are from EP to EN for R, from EN to EP for L and from R to L for S. Thus, when comparing flows to the septum (S) of the middle cardiac section (S_2) during $+3G_Z$, the middle portion (M) has 1.45 and the left septal portion (L) has 2.10 the flow to the right septal portion (R).

R = Right Ventricle
L = Left Ventricle
EP = Epicardium
EN = Endocardium
S = Septum
M = Middle
1 = Basilar 3rd
2 = Middle 3rd
3 = Apical 3rd

DISCUSSION

CHISUM (United States): With reference to the sharp drop in blood flow to the eye, though evidence of consciousness is present, does it not seem reasonable to assume that blackout had occurred?

HAMLIN: Absolutely.

BRENNAN (United Kingdom): Did the intra-ocular pressure of the animals vary during the experiment? This may well influence blood flow to the retina and choroid.

HAMLIN: I do not know if intra-ocular pressure changed.

GILLINGHAM (United States): Could you please comment on the relation between the increase in myocardial perfusion which you noticed at high G loads and what you would expect to be the increase in myocardial oxygen demand necessitated by the high G environment?

HAMLIN: I presume the dominant proportion of cardiac output perfused the heart, but whether or not that blood contained adequate oxygen due to $\dot{V}/\dot{Q}$ abnormality is equivocal.

UTILIZATION OF HUMAN CENTRIFUGE FOR TRAINING MILITARY PILOTS IN THE EXECUTION OF PROTECTIVE STRAINING MANEUVERS

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ABSTRACT

The present research aimed to investigate on the importance of the utilization of human centrifuges in the training of pilots in a rational execution of protective straining maneuvers.

With this scope, a group of young military pilots, of the Italian Air Force,were submitted to $+G_z$ for comparatively long durations.

During the first centrifuge run the subjects were instructed to refrain from performing any voluntary straining maneuvers.

Later, the same subjects were submitted to the same acceleration pattern, accompanied, this time, by the execution of the aforesaid straining maneuvers. Exposures to G were repeated.

Changes in performance and in tolerance to G were evaluated by recording morphological changes of EKG and heart rate. Besides, subjective feelings of pilots were recorded.

INTRODUCTION: We intended, with the present work, to appreciate the importance of a correct learning and execution of the M-1 anti-g maneuver (mainly consisting, as it is universally known, in protracted expiratory acts - about 5 or 6 per minute - executed against the resistance offered by half-closed glottis, with the intermission of very short inspiration, "gasps").

MATERIALS AND METHODS: On this premises we conducted a survey on 18 Italian military pilots (Officers, aged 22-43: mean age 26,3 $\pm$ 5,0), each one endowed with a satisfactory experience of acceleration in flight.

First of all they had to learn the correct execution of the M-1 maneuver. Subsequently they were submitted to the following accelerative pattern in the human centrifuge of Italian Air Force Aerospace Medical Center:
from $+1G_z$ to $+3G_z$ in 30 sec (onset 0,06 G/sec), 15 sec at 3G, then 20 sec from 3 to 4G, 4G were sustained for 15 sec.

The experiment was divided in two parts, carried on at one day interval. In the first day the aforesaid pattern was executed three times, with 2 minutes intervals, the first time in absence of any voluntary anti-g maneuver, and with skeletal muscles as much relaxed as possible, and the third time protecting oneself by means of the M-1 maneuver, while during the second run subjects were free to behave at will, that is to say executing any anti-g maneuver they were accustomed to.

The onset of protective maneuvers had been fixed at the reaching of 3G, they had to be continued till the subsequent deceleration.

Next phase - carried on the following day - was characterized by the fact that the subjects repeated and identical sequence of experiments, wearing, this time, an anti-g suit.

Just before the centrifuge run, and during G-exposure, electrocardiogram was recorded.

Later the subjects were invited to fill in a questionnaire, concerning their previous experience on G and G-tolerance, and the subjective consequences of the execution of anti-g maneuvers in any considered condition.

RESULTS: From the examination of the questionnaires, we observe that:

1) When a student everybody had learned from his instructors some non completely standardized anti-g maneuvers, consisting in straining skeletal muscles (and in particular those of the legs and abdomen). More or less conscious apnoea was maintained for long periods during G-exposure, although only two subjects did mention it expressely. These maneuvers were currently applied in flight.

2) In regard to M-1 maneuver, only 2 subjects had a previous knowledge of it. None, however, had ever tried it.

3) 10 subjects affirmed that learning the M-1 maneuver did not offer any particular difficulty, while the remaining 8 failed to give any direct answer to this question.

4) 5 said the execution of the maneuver under G-stress is more difficult, and that this difficulty is enhanced by the inflation of the anti-G suit.

5) 11 pilots complained of respiratory troubles (cough, throath irritation) during the expiratory phase of M-1. Two of them experienced also a mild dizziness.

6) In regard to protective effects of M-1 maneuver, only 5 admitted that this maneuver, although a little uneasy, may afford a certain improvement in tolerance to acceleration - better than with "spontaneous" maneuvers adopted by them. On the other hand, 2 affirmed that M-1 could cause a certain worsening of general subjective conditions. The remaining ones failed to observe any difference between the protective effects afforded by M-1 and by "spontaneous" maneuvers.

So much for subjective feelings.

The objective observations that follow rely on the examination of EKG recordings:

1) In table I we report heart rate (HR). One can observe a consistent increase of HR under accelerative stress, till 180 beats per minute in one subject, more relevant during the execution of M-1 maneuvers. Of course, the use of anti-g suit caused a comparative reduction of HR.

TABLE ONE - HEART RATE BEHAVIOR

At rest 91 ± 18

		relaxed	"spontaneous maneuvers"	M-1
WITHOUT ANTI-G SUIT				
1.5 G		101±30	115±26	118±26
2 G		114 21	121 19	118 21
3 G		124 19	131 15	131 20
4 G	0 sec	133 19	136 18	147 19
	5 sec	136 19	140 14	145 22
	10 sec	137 20	138 22	144 25
	15 sec	135 21	140 20	145 23
WITH ANTI-G SUIT				
1.5 G	------	------	------	92 31
2 G		114 18	109 9	112 18
3 G		105 19	113 21	117 21
4 G	0 sec	109 20	123 23	127 20
	5 sec	112 21	120 22	130 28
	10 sec	112 20	119 19	134 25
	15 sec	105 22	124 19	131 26

2) Let us consider, now, some morphologic features in the EKG.

a) At least 5 cases showed fairly evident troubles of repolarization phase. A certain improvement was obtained thanks to anti-g maneuvers (both "spontaneous" and M-1). Such improvement can be explained, in some instances, by the slowing of HR, although, in many cases, such an improvement is not depending from HR, being present even in spite of a further increase of HR.

b) In 6 other cases we observed an improvement of EKG-recording (consisting in a higher voltage of T wave) during the execution of both anti-g maneuvers.

c) In the remaining cases we failed to put into evidence important changes of EKG in relation to anti-g maneuvers.

d) In one case there was a loss of consciousness of short duration, with remarkable bradycardia with intermittent ventricular extrasystoles.

CONCLUSIONS: In conclusion we can say:

1) All the various experimental conditions were characterized by some changes in HR, consisting in a progressive and discontinuous increase, provoked, of course, by accelerative stress, but also connected to psychological conditions.

2) Anti-g maneuvers caused in many cases (not less than 13) a useful effect both with and without anti-g suit (independently from HR behavior).

We were not able, however, to put into evidence a consistently higher efficiency of M-1 maneuver if compared to "spontaneous" maneuvers. An outstanding role was played, beyond any doubt, by psychological factors related to the execution of an unusual task in particularly stressing conditions.

Indeed not less than fifty per cent of pilots expressed their worry concerning the possibility of a correct execution of the M-1 maneuver without seriously interferring with control in flight.

This notwithstanding, we feel authorized to observe that M-1 maneuver is comparatively well tolerated and that, in some cases, it gives satisfactory results, objectively assessed.

We are sure that, if student pilots were conveniently trained in the execution of M-1 maneuver (which is, in our opinion, to be preferred in case of long sustained G), satisfactory results could be reached, both from objective and subjective standpoints.

THE USE OF A FIXED BASE SIMULATOR AS A TRAINING DEVICE FOR HIGH SUSTAINED OR ACM (AIR COMBAT MANEUVERING) $+G_z$ STRESS

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SUMMARY

The use of a fixed base simulator (human centrifuge) has been used in aviation medicine research since approximately 1936 when the Luftwaffe constructed their centrifuge in Berlin. However the use of a centrifuge as a training device to improve $+G_z$ tolerance had not been instituted until the initiation of this program at USAFSAM. This report deals with the imposition of $+G_z$ stress on 92 highly experienced Tactical Air Command fighter pilots. A typical class of 22 of these pilots had an average (± SEM) of 1351.66 fighter hours (± 158.82), and were 29.04 years of age (± 0.54). In this same class of 22 fighter pilots they estimated the highest G that they had ever pulled was ± 9.0 G_z for 6.4 sec (± 0.17/± 1.23). From this data it was apparent that fighter pilots flying the F4E Phantom jet did not pull high sustained G as described at USAFSAM (> 6 G/15 sec). Therefore a centrifuge program was initiated in order to train pilots at high sustained G and at ACM G. The profile used was as follows: (a) +3 G_z/15 sec--this was an orientation run in order to familiarize the pilot with the centrifuge environment. (b) +5 G_z/45 sec--this extended run was designed to enable the pilot to learn to pace his breathing and straining maneuver properly while being exposed to G sufficient to cause the anti-G suit to inflate. (c) A final ACM type profile that exposed him initially to +5 G_z/10 sec and then proceeded to +8 G_z/30 sec, decelerated to +5 G_z/10 sec and finally the centrifuge was brought to a halt. Under these conditions the 92 pilots' heart rate and rhythm was continuously monitored. Resting heart rate for this larger group prior to initiation of the run averaged 115.98 bpm (± 1.64). While the maximum heart rate at +8 G_z was 167.04 bpm (± 2.12) in all instances using a students t-test, the P value is < .001 when the heart rates at any G level are compared to the pre-run control heart rates. However the important aspect of this training episode was the fact that all of the pilots were able to complete the proposed series of runs after receiving training by the centrifuge group at USAFSAM without a loss of vision. A final quote from one of the pilots is appropriate in this instance and is as follows: "I think it is an invaluable experience here to get the G instruction and then get in the centrifuge and have qualified physicians and physiologists critique you, your techniques, your methods, while you are actually under the G stress." Subsequent to these runs TAC Flight Surgeons and Physiological Training Officers have also undergone a similar G training experience.

INTRODUCTION

The use of human centrifuge as an investigative tool for determining blackout thresholds in experimental subjects and in aircrew members has been common practice in modern aviation medicine since the construction of a centrifuge in Berlin in 1936 (1). Prior to this date other centrifuges had been constructed but were used for a variety of purposes, including the treatment of mentally ill patients as well as for quantitating experimental animal response to various G stresses (2). During W.W.II human centrifuges were designed and constructed for the purposes of developing, testing, and prototyping anti-G protective devices such as suits and valves. The Mayo Clinic centrifuge, constructed in 1942, was almost totally dedicated to studies involving the physiology of acceleration although it was also from this laboratory and from Wright Field that the USAF G3A anti G suit was developed in 1944 and used successfully during the latter stages of W.W.II. The RCAF centrifuge, placed in operation in 1940, was used not only as a research device but also as a means for evaluating a hydrostatic anti G suit developed by Dr. W. F. Franks and used by England in the early years of the war. The second Wright Field centrifuge was placed in operation in 1943 and, between Wright Field and the Mayo Clinic, experimental anti-G suits and valves were tested on human subjects.

During the post W.W.II era a number of centrifuges were designed for specific purposes. The US Navy constructed the most sophisticated centrifuge in the world in 1950 at Warminster, Pennsylvania. In its early years it was used primarily to determine changes in human operator task performance while pilot subjects were exposed to either $+G_z$ or $+G_x$ forces. However, with the onset of the NASA space program this centrifuge proved its true worth as a training device. The early Mercury astronauts trained in a high G environment on this centrifuge, learning what could be done by an astronaut when exposed to $+G_x$ (chest to back) forces as high as 11-12 G, simulating either the Mercury launch or re-entry G profiles. In 1962 astronaut D. K. Slayton stated "we feel that the centrifuge has been one of our most valuable training devices." With these prophetic words a new era in the use of centrifuges commenced. A large centrifuge was constructed at the Lyndon B. Johnson Space Center, Houston, Texas in 1965 that was specifically designed to familiarize U.S. Astronauts with G forces similar to those that would be obtained during the launch and re-entry of the Gemini, Apollo, Skylab and Space Shuttle capsules. For its use as a training device in the Apollo program, the floor of the centrifuge room in the Flight Acceleration Facility was modified to simulate the moon's surface. The main arm of the centrifuge served as a means to suspend the astronaut so that his weight was 1/6 his Earth weight, thus simulating the difficulties of locomotion on the moon. The centrifuge main arm moved about the room at a walking speed thus the suspended astronaut trained at "1/6 G".

The Russians also use centrifuges to train their cosmonauts to simulate launch and re-entry G forces. According to Kotovoskaya (3), cosmonauts were not exposed to the same G levels used during human experiments on the centrifuge (+9.5 - 13.0 G_x) but she stated that "cosmonauts must be prepared for (trained for) re-entry G forces much greater than normal if an emergency re-entry is required."

The Japanese Air Self Defense Force centrifuge at Tachikawa, Japan is expanding its utility by serving as a training simulator for aircrew members (4). Also the German Air Force centrifuge at Fürstenfeldbruck is being modified for use as a high G training device for pilots selected to fly the multi-role combat aircraft (MRCA). Finally, the centrifuge at the Karolinska Institute, Stockholm,

Sweden is being used to train fighter pilots of the Swedish Air Force, in addition to its use as an excellent research simulator.

This then serves as a brief review of other centrifuges and their employment as training devices. We will now focus attention on the various types of training episodes in which the U.S. Air Force School of Aerospace Medicine (USAFSAM) human centrifuge has been used since its construction in 1963 (Fig. 1).

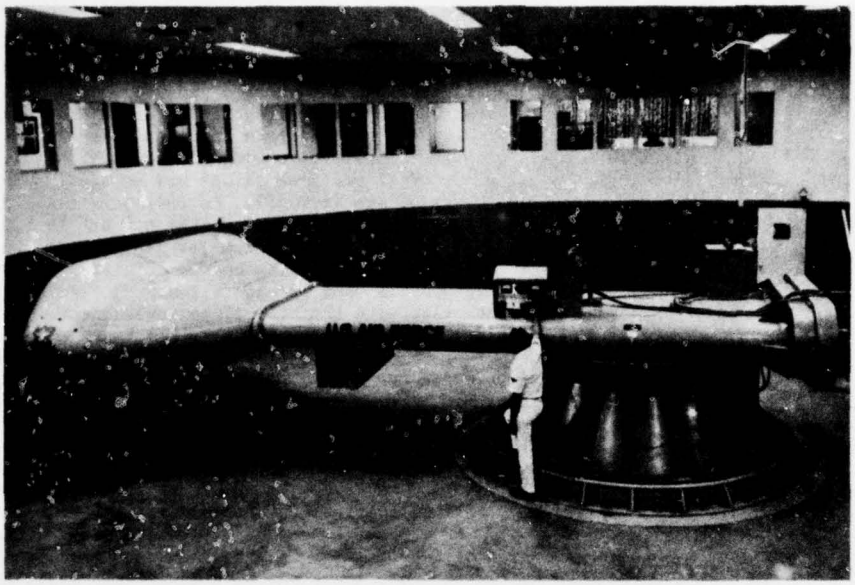

Figure 1. USAF School of Aerospace Medicine human centrifuge.

This electro-hydraulically driven machine has a 20 ft radius arm, maximum of 94 rpm (50 G) with a 600 lb payload. The interior of the gondola can be mocked up as a fighter aircraft cockpit with throttle controls and center positioned stick or a side stick controller. Various techniques have been employed to simulate closed-loop control through the pilot operator in the gondola, thus improving its image as an inflight simulator. This centrifuge was first used as a training device by student pilots from the Aerospace Research Pilots School (ARPS), Edwards Air Force Base, California beginning in 1964. For several years thereafter, being a graduate of the School was a prerequisite for consideration by NASA as an astronaut for their various space programs, by the Air Force for their Manned Orbiting Laboratory program and by the Air Force as a test pilot in the X-15 program. During a 3-week period, student pilots enrolled in the ARPS came to Brooks AFB and attended a course titled "Bioastronautics for Space Research Pilots (ESRP)." An important segment of the course included intensive centrifuge training that involved (a) exposure to $+G_x$ forces simulating those that would occur during launch and re-entry of a space vehicle and (b) the $+G_x$ forces involved in escaping from an exploding Saturn C-V fuel cell on the launch pad using the escape tower (5). Over 240 ARPS pilots were trained on the SAM centrifuge before the course was phased out at Edwards AFB.

Since all U.S. Air Force Aerospace Medicine training, Physiological training, and Aerospace Nursing training takes place at Brooks Air Force Base, it followed that centrifuge indoctrination, the physiology of high $+G_z$, and Air Combat Maneuvering G forces training would become an integral part of their medical training. As an example, during their second year, Residents in Aerospace Medicine receive 6 hours of lecture and 16 hours of centrifuge training. Going one step further, the Surgeon General of the Tactical Air Command requested that all of his Flight Surgeons and Physiological Training officers assigned to fighter wings receive an intensive course - including centrifuge training - in acceleration physiology at Brooks AFB. A G versus time profile of their training is shown in Fig. 2. With non-pilots the two 8 peaks are reduced to +7.0 G_z or less, depending on their tolerance to prior runs.

Finally, in 1971 the Commander, Tactical Air Command, approved high G training for student pilots assigned to the Fighter Weapons Instructor Course, Fighter Weapons Center, Nellis Air Force Base, Nevada. This training involved lecture, demonstration, and lastly exposure to high sustained $+G_z$ forces, simulating maneuvering G forces in F4E aircraft (Fig. 3).

METHODS

Ninety F4E fighter pilots (or navigators) and two flight surgeons were exposed to high, sustained $+G_z$ forces (> 6 G for periods lasting longer than 15 seconds) (6) on the USAFSAM human centrifuge. The pilots wore their own personal flight boots, helmet, oxygen mask, anti-G suit and flight suit. They breathed ambient air and had their anti-G suits fitted to their comfort and as they were used during air combat maneuvering (ACM) in F4E aircraft, which was also the fighter aircraft in which they held their current aeronautical rating. Several of the individuals being trained on the centrifuge were navigators rather than pilots (or two flight surgeons) but all were experienced in the G forces involved in F4E ACM and, for the purposes of this report, will be grouped together. Vital statistics from one class of 21 pilots is as follows:

TABLE I (± SEM)

Age (yrs)	Height (in)	Weight (lbs)	Total Flying Time (hrs)	Fighter Time (hrs)	Highest $+G_z$/Time (G/seconds)
29.0 ± 0.5	71.4 ± 0.6	181.6 ± 4.0	1633 ± 163.5	1351.7 ± 158.8	9.0/6.5 ± 0.2/1.2

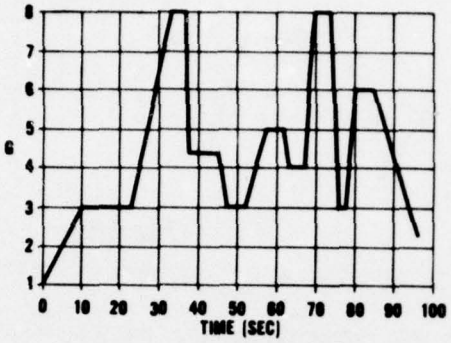

Figure 2. A reconstructed F4E, G versus time air combat maneuvering profile.

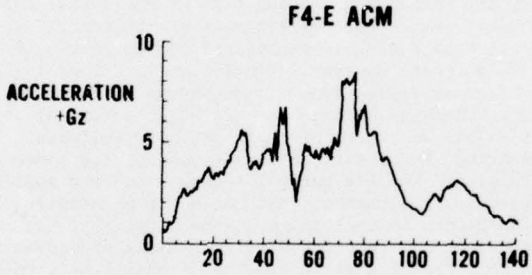

Figure 3. An accelerometer recorded G versus time air combat maneuvering profile taken from an engagement between two F4E's. A modification of ACM traces such as this was used in the training. The abscissa is time in seconds.

The pilots represented in Table I are a highly experienced group of fighter pilots who are moving upward in their flying and command career in the Air Force. It is interesting to note that when asked, "What was the highest $+G_z$ maneuver they had ever pulled and for how long?" the average was +9.0 G_z for 6.47 seconds (last column). This is indeed a high, sustained G maneuver in the F4E since this aircraft will lose altitude and airspeed at these G extremes.

All of the pilots presented themselves with a completed Class II flying physical examination. A brief medical history was taken by the attending physician and a physical examination was undertaken if he deemed it necessary. After a two hour lecture on the physiology of high, sustained $+G_z$ forces the pilots commenced their centrifuge training. They were instrumented for two leads of ECG, sternal and biaxillary. These were used for detecting changes in heart rate and rhythm and were not calibrated for clinical quality ECG. A closed circuit TV was used for visual monitoring, while continuous voice communication was maintained by a hot microphone between the pilot in the gondola and medical personnel in the recording room located upstairs. Data were recorded on both a Sangamo Model 700 14 channel magnetic tape recorder and an 8 channel Brush Mark 200 strip chart recorder. They included G forces, ECG, instantaneous heart rate derived from the ECG, G suit inflation pressure and tracking task performance (7). The G profiles used during this training are shown in Figure 4. Following familiarization with the cockpit, the pilot is instructed again on the proper method of performing the M-1 or L-1 straining maneuver and how to pace his breathing during the high G exposure (8). Following this he has his first centrifuge ride, +3 G_z for 15 seconds. This allows him to experience relatively low G forces below normal blackout threshold but great enough to allow G suit inflation. The second exposure is +5 G_z for 45 seconds. This is usually the most prolonged G exposure he has ever experienced and is specifically used to familiarize him with a breathing pattern that will be used in the subsequent high G exposure. The natural tendency is to hyperventilate and this dangerous tendency must be judiciously avoided, particularly in a high G environment. The end result could be a catastrophic episode of unconsciousness. The third and final run is a step function exposure going to +5 G_z/5 seconds and thence to +8.0 G_z for 30 seconds, back to +5 G_z for another 5 seconds and finally to a halt. After being removed from the gondola, the subject is immediately debriefed by either the flight surgeon or physician monitoring the run or by a physiologist observing the run. A video tape of his face and a simultaneous voice track are played back to demonstrate his method and degree of success in performing the straining maneuver during high G. An evaluation of his heart rate and rhythm during G are presented from the strip

chart record. Finally, he is questioned about his inflight F4E G experiences and these are related to his centrifuge G training. He then observes other classmates going through an identical experience and - in this instance - the TV monitor and video tape playback systems are important, particularly when a pilot performs an improper straining maneuver or fails to pace his respiratory pattern adequately.

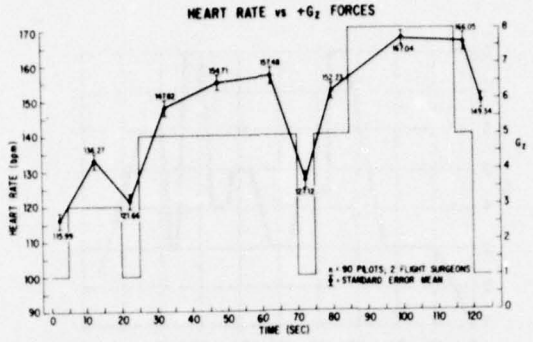

Figure 4. Pilot heart rate versus $+G_z$ forces. The light line represents the $+G_z$ profiles used to train pilots. At least 5 minutes rest occurred at the 1 G control points.

RESULTS AND DISCUSSION

A determination of whether centrifuge training is beneficial and improves a pilot's ability to tolerate high sustained G during air combat maneuvering remains subjective and difficult to answer. It would be beneficial if a pilot could state that "as a result of the centrifuge training I was able to outmaneuver threat aircraft better than during an equivalent period last year before receiving the training." It is not possible to do this, however. Simulations of other inflight phenomena during pilot training are also laced with subjective impressions of the student pilot. For example, during undergraduate pilot training, inflight maneuvers that cause vestibular or visual illusions or disorientations are demonstrated to the student pilots as a part of their training syllabus. Air Training Command would like to have some means of evaluating the benefit of this training, i.e., how did it help the student once he graduated from pilot training? Was his natural tendency to be disoriented suppressed as a result of the training? These are subjective evaluations that can never be answered except on an individual basis. Centrifuge training then appears to fall in this same category. You can very readily discuss the effects of high G forces or one can demonstrate these effects using pilots as their own subjects - as in this study - but the benefit of this training still remains in an ill defined category of success. It has even been suggested from time to time that the centrifuge become a part of the pilot training program to demonstrate the effects of high G forces or maneuvering G forces on pilots flying air superiority weapons system. The logistics of such a suggestion does not make it feasible for all undergraduate pilot trainees to undergo this type of stress on a centrifuge. However, by selecting the pilot trainees and restricting those selected to potential pilots of high performance fighter attack aircraft it becomes more feasible to initiate a centrifuge training program. The student pilots who are reported herein were assigned to the Tactical Fighter Weapons School, Nellis Air Force Base, Nevada. The course they were enrolled in was conducted 3 times per year, 25 students per class, thus it became logistically possible to train them on the USAFSAM centrifuge without interrupting on-going research programs.

The most important person evaluating the results of the G training remains the individual fighter pilot himself. One of the highly skilled pilots who had never been exposed to high sustained $+G_z$ forces before stated "I feel every pilot attending the Fighter Weapons Instructor Course in the Tactical Air Command should receive the high G training at Brooks Air Force Base." Another stated "I think it is an invaluable experience here to get the G instruction and then get in the centrifuge and actually have qualified physicians and physiologists critique you - diagnose your straining methods - while you are actually under the G stress." Many have stated that in the flying environment and while engaging threat aircraft they are not aware of the increased G force or of their individual methods for controlling the effects of these G forces. The stress and the flying requirements on the pilot make it impossible for him to concentrate on this particular area. However, since he is a captive audience on the centrifuge, he can evaluate the effects of G and can be shown the errors inherent in his method of straining against the high G forces. To this end the process of learning is greatly contracted in time and the pilots who have undergone this training feel that they have increased their confidence in the body's ability to withstand high G forces during an engagement with threat aircraft.

Physiologically the information recorded from the subject during training was heart rate and rhythm derived from the electrocardiogram trace. Figure 4 is a graph charting the heart rate of the 92 pilots involved in the study in conjunction with the G profile. High resting heart rates can be seen before each run and the resting heart rate increases with subsequent runs. For example, the average resting heart rate for the +3 G_z episode is almost 116 bpm whereas it is 122 and 127 bpm respectively before the +5 G_z and +8 G_z peak runs. The increasing heart rate in the +5 G_z run is significantly greater than the resting heart rate before the +3 G_z run ($P < .001$). Also the increased heart rate in the +8 G_z run when compared to its pre-run control value is highly significant ($P < .001$). In some individual responses heart rates approached but did not exceed 200 bpm during the +8 G_z, 30 sec epoch. A heart rate of this magnitude - achieved by one pilot - is the rate at which a run is stopped by the medical monitor. The high resting heart rates in these pilots is probably their normal response to an environment unfamiliar to them, i.e., the gondola of the centrifuge is not the cockpit of an F4E aircraft. It has been shown in previous studies that the resting heart rate of these fighter pilots is less in the cockpit environment of an F4E aircraft than it is with similar pilots exposed to G on the centrifuge. For

example, F4E pilots' heart rates during the roll out before takeoff averaged approximately 97 bpm (9) compared to the trained pilot in the SAM centrifuge who had control resting heart rates of 116 bpm or more (this study). Another factor that may cause a high heart rate could be the resting catecholamines in the particular group of pilots. Burton's study recently indicated that fighter pilots flying high perfomance aircraft on a daily basis in an ACM environment have resting catecholamine levels much greater than their fellow pilots who are not flying this type aircraft (10).

In one class of 25 student pilots (part of the G_z subjects), cardiac arrhythmias were noted during the runs and the number of arrhythmic beats increased as the G level increased. The number was also increased with time at G. The cardiac irregularities included numerous episodes of multifocal atrial premature contractions, multifocal ventricular contractions and in one individual a run of ventricular tachycardia at +5 G_z. Only one of the arrhythmias caused sufficient concern to stop the run and this was the ventricular tachycardia at +5 G_z. Out of the 25 pilots, 12 exhibited premature ventricular contractions (PVC's) during G while only 6 demonstrated premature atrial contractions (PAC's). Two of the pilots had runs of ventricular tachycardia, one during the 5 G/45 second exposure with sufficient number to preclude his continuing on the final ride to +8 G_z/30 seconds. Subjectively, many of the pilots reported blurring of vision, intermittent loss of peripheral lights and central dimming to the point of tunnel vision. Although all pilots had positive control over the centrifuge and could stop by releasing a depressed switch on the throttle, none elected to do so because of a loss of vision. They were aware of prompting by the central observer as vision began to fail. As they increased muscular effort during the straining maneuver, vision improved and in most instances returned to normal while at the most severe G loading.

Petechiasis was noted in unsupported peripheral areas such as the volar region of the hands or areas of the calf or ankle region in which counter pressure was not applied during anti G suit inflation. None expressed any discomfort during the high G exposure due to the hydrostatic column length from the heart to the ankle except for one pilot who had previously experienced a compound fracture of the leg just below the knee joint. This severe pain in the region of the break was relieved as soon as the high G forces were released during deceleration of the centrifuge. He was also short and retracted his feet back against the seat (unknown to the observer) and thus did not gain benefit from pushing his feet against the rudder pedals. If he had done this, the possibility exists that the pain in the lower legs might not have been as severe.

The most difficult thing for the pilots to achieve during their exposures to high sustained G was the timing of the breathing/straining/G suit inflation cycle. Many had an initial tendency to hold their breath while straining for extended periods of time and only broke the breathholding episode after 7-10 seconds in order to take another quick breath. Others tended to hyperventilate, breathing every 2-3 seconds. A properly executed M-1 or L-1 straining maneuver should be repeated every 4-5 seconds while a pilot is pulling high +G_z. A respiratory cycle during G commences with a quick inspiration as the anti-G suit inflates, followed by a forceful expiration against a closed or partially closed glottis. At the same time the pilot strains maximally against the inflated G suit and this effort is then followed by another quick inspiration. The above cycle is then repeated. The inspiratory part of the cycle should be a quick gasp in order to avoid a prolonged fall in head level arterial blood pressure. Once the pilots become familiar with this routine it is quite easy for them to maintain normal vision. Most stated that they had never been trained adequately in the performance of the M-1 or L-1 straining maneuver and certainly none had been taught to pace their breathing properly during sustained G (since none had been exposed to high sustained G before). Cine film of the pilot's performance of a forceful expiration showed the MBU5/P oxygen mask being pushed out and then dropping down with each breath. A picture of a pilot at +4.5 G_z is compared to the same individual at almost +8.0 G_z (Fig. 5a and 5b).

Figure 5a. Pilot's face at +4.5 G_z. Very slight facial distortion.

In the final high G profile the initial +5 G_z step allows the pilot to prepare for the +8 G_z peak both by straining, by having the anti G suit inflate, and by reflex compensatory mechanisms responding to the +G_z forces. In addition, this profile would also be similar to a fighter pilot's response when

first commencing to engage a threat aircraft, i.e., pulling a +5 G_z turn in order to gain visual or positional advantage thence pulling harder G in order to get a target in gun range.

Figure 5b. Pilot's face at +7.5 G_z. Severe facial distortion and oxygen mask slippage.

The advantages of having experienced pilots receive this high G training on the centrifuge appear to outweigh the disadvantages of taking these individuals away from their busy operational schedules in order to receive the training. With these particular tactical fighter pilots, the centrifuge was operated over the weekend in order to avoid conflicts with their pre-established weekday schedules. Several of the pilots noted some degree of disorientation following the high G exposure on the centrifuge; therefore none was allowed to fly as primary pilot for the remainder of the day. In all instances the entire class returned to Nellis AFB the day following their high G rides on the centrifuge.

If airframe manufacturers are capable of designing fighter aircraft that will allow high sustained G maneuvering as part of the performance envelope of the aircraft then pilots flying these aircraft must be trained to withstand and protect themselves against these high sustained $+G_z$ forces. Any compromise of vision or consciousness compromises the success of an air battle and should not become an acceptable part of planned attrition. It is not possible to estimate the number of pilots or aircraft that could be saved as a result of high G training. However, it is reasonable to believe that high G training will improve a pilot's ability to survive an engagement against an equivalent threat aircraft.

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The research reported in this paper was conducted by personnel of the Environmental Sciences Division, Biodynamics Branch, USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, United States Air Force, Brooks AFB, Texas.

STRESS RESPONSE AND STRESS TOLERANCE IN FIGHTER PILOTS DURING 6 G MANOEUVRES

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The EEG Research Institute, Gaustad, Oslo 3.

Summary: EEG and EKG has been monitored from 250 active fighter pilots flying combat training involving repeated 6 G turns and pullups. 50 students and none pilots has been monitored while riding in the back seat of two seater fighters going through the same manoeuvres.

More than half of those pilots who had committed pilots error were unconscious with convulsions following 6 G manoeuvres. Gross EEG changes were seen in the EEG.

Studies of the EKG and heart rate illustrates the cardio-vascular response.

The well suited pilots has a quick response with increase of heart rate when needed to maintain adequate blood supply to the brain. The unsuited groups demonstrate a slow, unsufficient cardiac response leading to brain-anoxia unconsciousness and convulsion.

The student pilots and the none pilots fell in the same two different categories indicating a basic difference in the functioning of the autonomic nerves system in these two groups.

From time immemorial man like all other living species has lived and developed in a steady interaction with his environment. Over the years traits have been developed and adjustments made to our changing environment to provide not only the needed material goods, but also security and love for his family.

What superfically appears to be stress and anxiety is really a needed stimulation to achievement and accomplishment resulting in a genuine feeling of well-being and satisfaction as the task is completed.

Man is in one way a homogeneous species, but by closer study we find there are marked differences in the way he is equipped physically and mentally. Man has a great capability for adaptation, just think in recent years how he has adapted to work on the ocean floor, to space travel and working missions on the moon.

As members of the human species we have all our strong and weak features. To the extent these strong and weak points are recognized. They are taken into account in our selection of our role in society.

Sometimes by mistake we seek occupations for which we are ill adapted. I will illustrate this later. Stimulation to achievement of goals to which we are ill adapted physically and mentally, inadvertently results in overload, in break-down and pathological behavior.

During performance of a task there is always a relationship between how involved the task is, how demanding it is both physically and mentally, and the energy the performer must put in to the task.

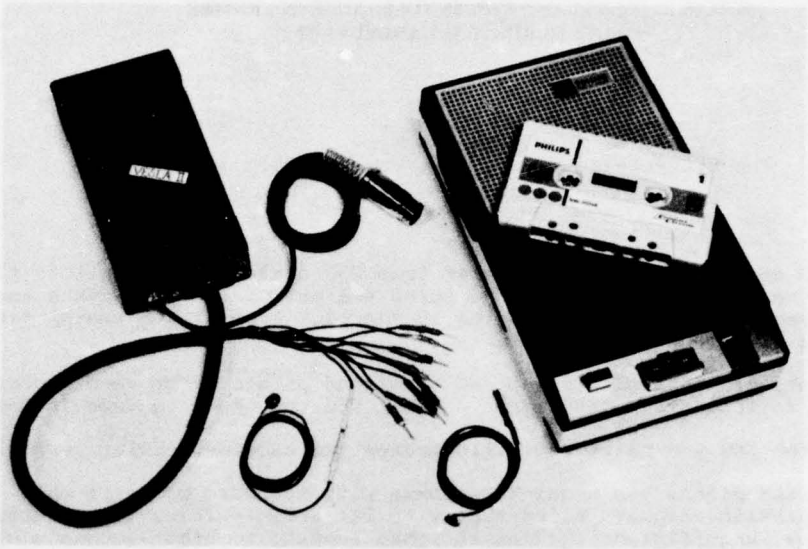
There is a relationship between the complexity of the task and the vigour and alertness required to perform the task satisfactory. The vigour and alertness come in response to the stimulation, the anxiety and stress created by the task.

It is further a common observation that tasks that appear to the performer dull and uninteresting are performed in a lax and even sloppy way which may be contributed to the lack of stress and stimulation.

At the EEG Research Institute we have for 20 years studied pilots, divers, rescue personell, radar operators and other subjects during the performance of lax and stressful tasks.

EEG, EKG, respiration, temperature and galvanic skin resistance are among the parameters monitored in more than 400 missions.

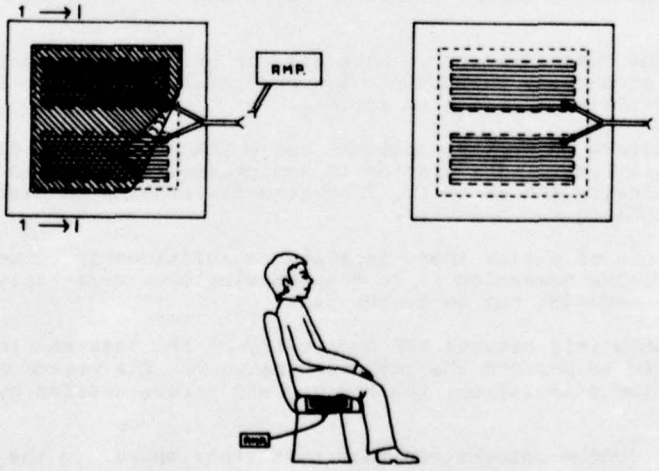
Over the years our equipment has been steadily improved from the first bulky, heavy equipment to the latest Vesla II Biomedical Monitoring Equipment which is carried in the subject's pocket, fig 1.



During the past 3 years we have developed the Vesla Seat Pad as a completely non-invasive way to study the subject's EKG during missions, The Vesla Seat Pad makes it possible to monitor EKG data through the subject's clothing without attachment to the subject of any sensors or leads whatsoever, fig. 2.

The subject simply sits, as illustrated on the figure, in his chair, or on his cockpit seat and the EKG is picked up continuosly from the minute he sits down through his clothing.

THE VESLA SEAT PAD



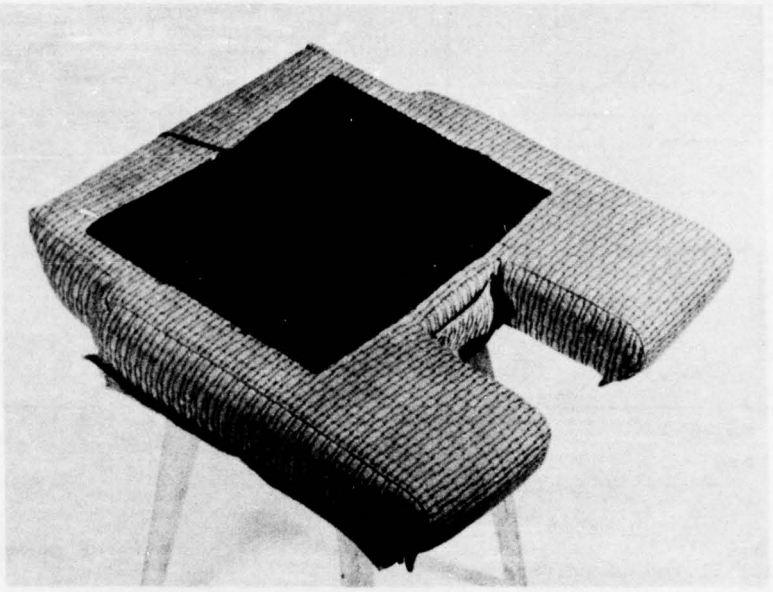
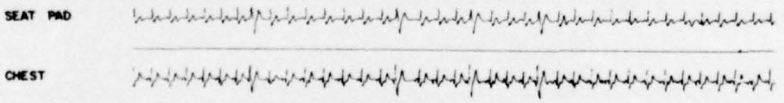


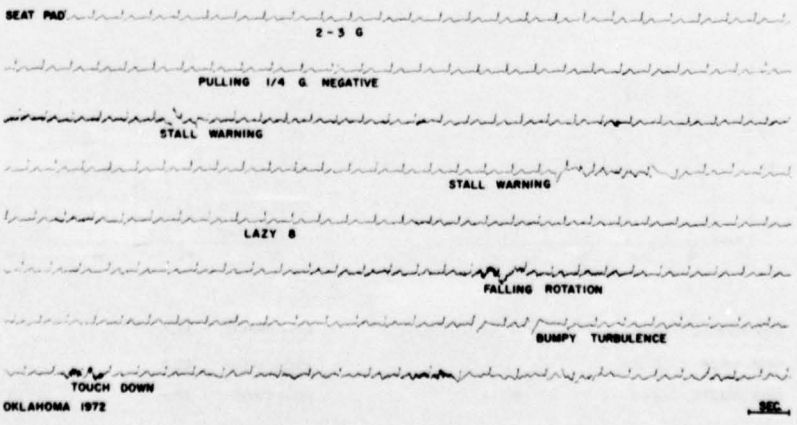
Fig. 3 is an illustration of the Seat Pad mounted on a DC-9 commercial airline cushion.

In 1972 we demonstrated the reliability of this technique for data gathering to the Federal Aviation Agency, in Oklahoma City, fig. 4 and 5.

AHRYTHMIC BEATS.



INFLIGHT MONITORING WITH VESLA EQUIPMENT AND SEAT PAD.



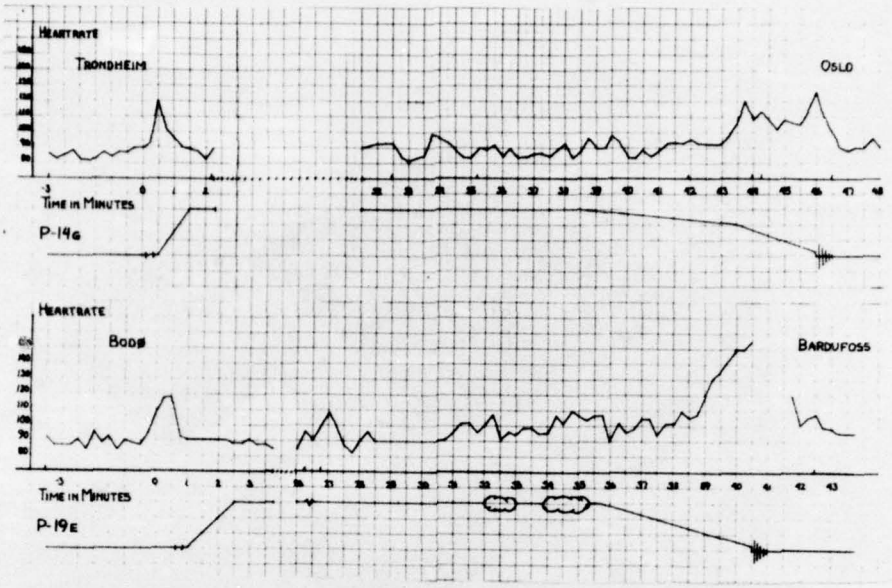
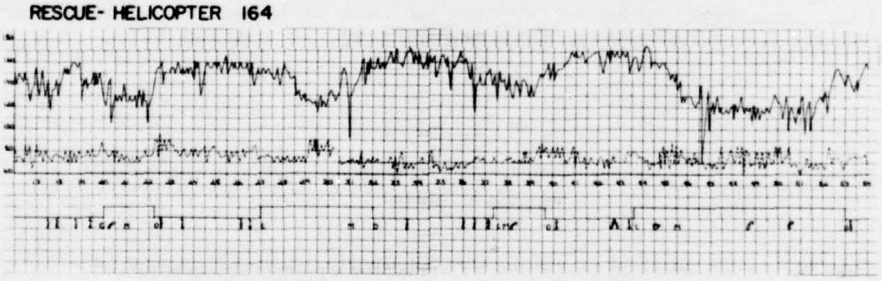


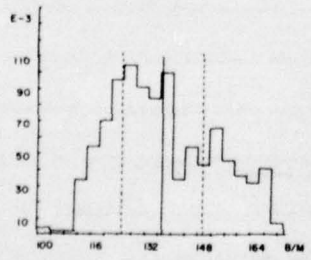
Fig. 6 illustrates the stimulation and workload response of commercial airline pilots flying to Bardufoss.

Fig. 7 and 8 illustrate through the EKG changes the challenge and complexity of a task for the pilot and the engineer during a rescue helicopter mission, and the response and the workload created by the mission.

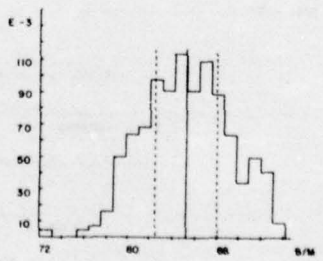


HEARTRATE OF HELI-ENGINEER HELI-PILOT DURING RESCUE MISSION

WORK-LOAD STUDIES



LOW FRAKTIL - 125,0
MEAN VALUE - 137,2
HIGH FRAKTIL - 149,3 150-3



LOW FRAKTIL - 82,8
MEAN VALUE - 85,6
HIGH FRAKTIL - 88,4 153-3

In our work we have steadily been able to see how certain situations stimulate the human subject and his autonomic nervous system. The blood flow and the heartrate are increased. During the demanding task the autonomic nervous system ensures that the brain has enough Oxygen and Glycose for efficient operation.

From 1958 to 1975 EEG and EKG has been monitored from 250 active fighter pilots flying combat training involving repeated 6 G turns and pullups. 50 students and none pilots has been monitored while riding in the back seat of two seater fighters going through the same manoeuvres.

In a study involving 55 randomly selected active fighter pilots it was demonstrated that more than half of those pilots who had committed pilots error were unconscious with convulsions following 6 G manoeuvres. Gross EEG changes were seen in the EEG.

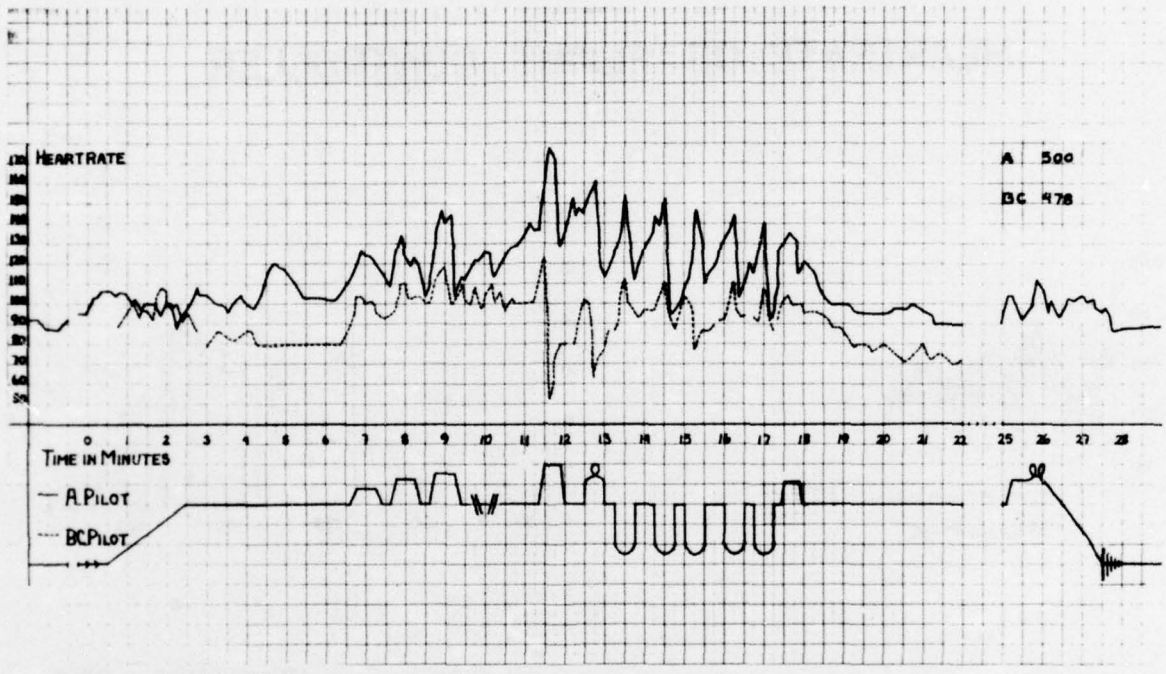


Fig. 9 illustrates the cardio-vascular response to stress in the two groups of pilots.

The figure shows the EKG/heartrate in two jet fighter pilots flying the same mission.

The well suited pilots has a quick response with increase of heartrate when needed to maintain adequate blood supply to the brain. The unsuited groups demonstrate a slow, unsufficient cardiac response leading to brain-anoxia unconsciousness and convulsion.

The student pilots and the none pilots fell in the same two different categories indicating a basic difference in the functioning of the autonomic nerves system in these two groups. An improved selection procedure will increase the efectivness of the Air Force and decrease the fatalities.

We are going to look at the movie: "Airborn Testing of Stress", from these flights, if time permits.

In the movie you will see how the stress of the task stimulated an excellent pilot. The pilot appeared well adapted, eager to do his task with all his knowledge, training and experience.

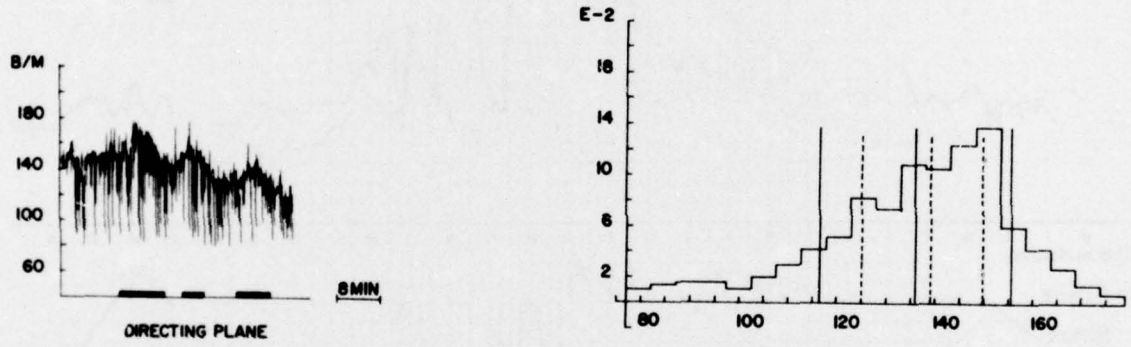
Those with a different type of autonomic nervous system with a slow response is quite obviously not suited for this type of missions and should select another occupation.

Years of experience with hundreds of pilots have demonstrated that it is not possible to alter the signature in the response of the autonomic nervous system laid down in all of us. This should be recognized and must be taken into account when we select our profession.

Note that the pilots who broke down during stress had a much lower heartrate than the pilot who worked efficiently.

Finally I would like to illustrate the heartrate response of radar flight controllers taking down planes in bad weather. These controllers are sitting in good chairs looking at the screen only pressing a few buttons and giving instructions. The task requires no physical work, but a high degree of alertness.

HEARTRATE OF FLIGHT CONTROLLER



LOW QUARTILE - 125,0

MEAN VALUE - 136,0

HIGH QUARTILE - 149,8

186-3

BODØ 1975

Fig. 10 is recording of the heartrate and workload of a controller while guiding 3 planes one after another. The response with the high heartrate ensures adequate supply of Oxygen and Glycose to the brain for effective function.

Conclusion: I have in this paper illustrated data from my material and shown how stress and anxiety may stimulate to achievement, followed by satisfaction and well-being. I have also illustrated how in som cases it may result in break-down of behaviour.

The data should make us more alert to the fact that although we are all equally needed - we are different built and equipeped, and this must be taken into account in our selection of our role in society. Thus the right man in the right job is a basis for a happy society.

ROUND TABLE DISCUSSION

CLARKE:
(United States)

In 1971, at Cocoa Beach, Florida, the subject of the pathophysiology of high G_z acceleration was discussed by an ad hoc committee of the U.S. National Academy of Sciences - National Research Council Committee on Hearing, Bioacoustics and Biomechanics. The committee was chaired by Dr. James P. Henry, well known for his pioneering work on high G, and included many senior scientists who had been or were at the time involved in research on this topic. I would like to ask Dr. Leverett to briefly review this meeting and its conclusions.

LEVERETT:

We at the U.S. Air Force School of Aerospace Medicine had recently (in 1970-71) completed a study involving human subjects exposed to forces up to +9 G_z or 45 seconds using either positive pressure breathing, the M-1/L-1 straining maneuver or the anti-G seat as adjuncts for improving tolerance. At the same time we began training TAC pilots on the centrifuge up to levels of +8 G_z for 30 seconds. Initial animal studies using the miniature swine as a human analog indicated some endocardial hemorrhage - particularly severe on the left side of the heart - at G levels similar to those used in human subjects with the RAF/IAM study designed to describe the physiological effects of high, sustained +G_z on subjects exposed to a maximum of +8G_z for 60 seconds. With all these studies as a background, it was decided to ask the advice of a group of acceleration experts to discuss the studies to date and determine, if possible, a G vs time history that would represent man's tolerance to these G forces. The meeting was composed of people from the following laboratories who were either currently engaged in acceleration research or had made significant contributions in past years:

USAF School of Aerospace Medicine (Brooks AFB, TX)
Aerospace Medical Research Laboratory (W-P AFB, Ohio)
Naval Air Development Center (Johnsville, PA)
Mayo Clinic (Rochester, MN)
State University of New York (Buffalo, NY)
Free University of West Berlin (Germany)
University of Southern California (Los Angeles, CA)
Aeromedical Research Laboratory (Bad Godesberg, Germany)
University of Miami (Ohio)
University of Michigan
Dept. of Transportation
Massachusetts Institute of Technology
NASA Langley (VA)

The high G studies from Brooks AFB were presented by several investigators and included identifying the high G pathology in swine, evaluating initial vectorcardiographic findings in humans indicating some possible mild myocardial hypoxia, and described methods to improve tolerance such as 5-7% CO_2 breathing, positive pressure breathing (30 mm Hg), and use of the M-1 and the L-1 straining maneuvers versus the Valsalva maneuver. The Mayo Clinic proposed some possible pulmonary damage at high G and this was substantiated by prior studies conducted by Dr. West. Arterial O_2 saturation was considered and it was felt that an imbalance in the ventilation-perfusion ratio existed at G and this imbalance worsened at higher levels of G, longer duration exposures and with the breathing of 100% O_2.

Several possibilities apparently exist to explain the endocardial hemorrhage at high +G_z. These could be (a) a decrease in end-systolic volume to the point that left intra-ventricular pressure exceeds aortic pressure and the empty ventricle beats against itself producing hemorrhage (b) a decrease in S_{aO2} to the point of myocardial hypoxia (c) a rapid heart rate (d) increased endogenous catecholamine acting on myocardium i.e., positive inotropic effect.

The two USAFSAM high sustained +G_z studies showed that man could tolerate these levels and maintain adequate vision. Also the arterial blood pressure levels at eye level were maintained adequately for cerebral perfusion. However arterial O_2 tension decreased with increasing G. Thus a point is reached where, in the conventional position, even intense muscular straining, positive pressure breathing, and the use of an anti G suit will not increase tolerance further. The limits of the experiments were +9 G_z/45 seconds and +8 G_z/60 seconds, respectively. At least in these two studies it was shown that a single sustained exposure was tolerable and acceptable in a properly motivated subject or pilot.

CLARKE:
(United States)

As described in the foreword, the speakers were asked to be prepared to discuss a series of operational questions posed to them by the chairman some 45 days in advance of the meeting. We will ask the speakers to now imagine you are addressing people with a military interest in the application of the kinds of data you have presented to this scientific group. We will try to see what kind of conclusions we can draw that are applicable to the operational situation; to state the areas of certainty as

CLARKE (cont'd.)

well as the uncertainty; and in general, to try to come as nearly as possible to some consensus opinion on the questions at hand. The questions are approximately in the order of urgency and importance, although all of them are certainly operationally significant.

We had a question this morning from the engineering community about what has changed since the days of the RAF Lightning as far as cockpit layout is concerned. Has the medical community impacted on any of these changes? What is the possibility that the tilt-back seat can offer some operationally significant advantage over the conventional seat? By operationally significant we mean one which not only allows us to take advantage of the physiological protection that is associated with assuming a more optimal position but one which also allows the pilot to continue to perform the functions that he is expected to perform. I hope that we will get at that question today as well as to get at the overall issue of operations in a high G aircraft. It is perhaps worth asking the engineering community a reverse question. "Is the high G environment really an environment that is useful from an operational standpoint?" If so, what kind of high G environment provides the best operational advantage? These kinds of questions have been addressed using simulations which I won't review for you since I think probably those of you who work in this area are thoroughly familiar with the work in progress. The general working hypothesis that we might take this afternoon is one based on ground base simulations and a limited number of closed-loop simulations on centrifuges, i.e., the guy who can pull the highest G is most apt to win the dogfight. If this is true, there are significant operational advantages to performing in this environment. The other thing one can say is that high levels of acceleration for short periods of time are operationally more significant than achieving very high levels of acceleration for sustained periods of time.

So, with this as background, we might address ourselves to the first of the questions that I posed in writing to this panel of experts some time ago.

The question is, "WHAT IS THE PATHOPHYSIOLOGIC RISK TO A TACTICAL FIGHTER PILOT EXPOSED TO AERIAL COMBAT ACCELERATIONS WITH DURATIONS ABOVE $+4G_z$ IN EXCESS OF 60 SECONDS WITH PEAKS OF 8 TO 10 G?" First with respect to single exposures; second with several exposures for several days; and third with regard to repeated exposures such as one might encounter in a career as a fighter pilot in the next generation of fighter aircraft.

I will try to very briefly summarize what I thought I heard as the answers and use that as the vehicle for opening up the discussion and perhaps coming toward a consensus and opinion. We started with Dr. Gillingham's paper in which subjects were exposed to a series of operational-like accelerations with quite high peak G loads. The conclusions that I believe he drew in general were that there were some evidences - EKG evidences - as well as biochemical evidences of changes in this subject population which were severe enough to cause concern and caution, but were not severe enough to keep us from stating that a pilot could on a one-time basis be reasonably safely exposed to that environment. We heard on the other hand experiments reported from Dr. MacKenzie and from Dr. Stone that pointed out the pattern of pathophysiology and biochemical changes that occur in the miniature swine. The significance of the blood flow changes, the endocardial, and myocardial hemorrhages that occurred and the myofibrillar degeneration that occurs were carefully qualified in the context of animal models with problems in terms of extrapolating between animal and man. But, nonetheless, the results indicated clear evidence for concern. The question to be answered is what is the degree of concern and the level of acceleration versus time at which this concern should be expressed in terms of the tactical pilot? I hope perhaps this will be discussed a bit more. The effects of back angle will be brought up at another question and addressed later. I believe with that we might ask the members of the panel to address themselves to this general question of what is the risk to a tactical fighter pilot exposed to a single episode of high sustained $+G_z$. Perhaps, Dr. Gillingham, you would like to start the discussion.

GILLINGHAM: (United States)

In the first place, I believe there was a question asked right after my presentation about whether or not the fighter pilots are different from our subjects; we have run a large number of fighter pilots and there is no doubt that they are very different. I don't know whether this occurs because of natural selection that results as these people develop into tactical fighter pilots or some other reason, but at least there is that difference between subjects and pilots. The pilots tolerate these G forces with considerably less difficulty. If any of you might have seen the movie that I showed at the Aerospace Medical Association meeting last year, we had a pilot that went up to 8 G peaks during the Lightweight fighter study doing his M-1 and he had essentially no problems at all. Now the High Acceleration Cockpit study which you recall went to a higher G level at different back angles was very stressful for the subjects. But one problem is that the subjects were also stressed with not being facile in the G environment. It was not as "old hat" to them as it would be to a fighter pilot who has thousands

of hours in a fighter plane and hundreds of hours in a maneuvering G environment. Another problem is that these subjects in the High Acceleration Cockpit study were instrumented with a radial arterial catheter and a number of other things. They were sitting in the gondola, perspiring, waiting for the run to start, anticipating in a very novel environment; and, I think that this may have contributed some to their less than adequate adaptation to this G environment. In addition to the fact that the fighter pilots have done well or better in general than our subjects, I think also that we should point out that there is a wide range of variation between subjects and also between fighter pilots. The subjects can vary widely in their straining tolerance. We had one subject Dr. Leverett has told me about which I didn't get to see who went to 9 G for 45 sec. without any particular ill effect as far as he was concerned. Now obviously I can't tell you what his heart looks like, but I don't believe he had any subendocardial hemorrhage; and he said that he could have gone to 10 G just as easily - and this is in the upright seat. And yet we have others that strain as hard as they can and they lose consciousness at 4 G. Perhaps with a little more training they would get to 5, 6, 7 but with great difficulty. I would propose that there would be an economic gain in selecting fighter pilots for high G work since the airplanes are becoming so expensive nowadays compared to what it costs to train a fighter pilot. Now with respect to the specific answers here, let's talk about a selected fighter pilot who is able to go to 7 G and just keep it up for an extended period of time (> 60 sec), which I am confident that many of these people who have the natural ability can do. With respect to single exposures, I don't think there would be any problem at all. We have people whose heart rates may reach 130 to 150 bpm during high G exposure. The mean heart rate was close to 170 bpm at about 8 G on fighter pilots who came through for high G training.

I don't think a single exposure of the type to which we are referring, namely an ACM with high peaks of 8 to 10 G would cause any problem at all. Several exposures per day, each day for several days would not cause a problem as long as they could rest after several days of this high G maneuvering. Also, I don't believe repeated exposure to ACM G forces encountered during a fighter pilot's career would lead to any physiologic damage. These ACM G profiles would have a duration of 60 sec with a mean G level of about 4 to 4½ and peaks of 8 to 10 G. This is the sort of ACM environment that would be expected with an airplane like the F-4E. Now the F-4E is not really capable of delivering the kind of accelerations that the F-15 and the F-16, the F-18 or the F-14 are able to deliver. And, the comment was made that it's good to go to a high G level for a short period of time to gain an advantage over your enemy. This is true between F-4E vs F-15 and the F-15 will always win because of its capability. But I'm afraid that the problem is going to come when you have an F-15 fighting an aircraft of equal capability in which case the average G level may be greater (around 6 or 7 G) for long periods because you are not going to be able to jump to 10 G and get an advantage on the enemy. You can jump to 10 G and he will jump to 10 G and you'll both be there for a while. I don't think we have the answer to the questions about whether these profiles will cause a significant pathophysiologic risk. I do believe to minimize the pathophysiologic risk of this kind of combat though that we have to select those pilots that seem to do best in that environment.

CLARKE:
(United States)

Dr. MacKenzie, what is the likelihood that the pathology seen in minature swine occurs, and to what degree might it occur in experimental subjects or pilots in high G_z environments?

MACKENZIE:
(United States)

The questions obviously do not have a simple answer. One must consider the conditions, under which the man will be exposed, at what age and the other environmental parameters. The lesion that we see in the pig is probably pleuri-causual and can be potentiated by so many factors that you have to know the pilot, i.e., you can't give an answer applicable to one pilot and apply it to another pilot. There will be a good bit of individual variation even in a selected population.

If there is a lesion produced in the heart of a pilot, he will be more susceptible to a second lesion within an hour or a day or two; and each time he is exposed and receives some additional injury he will become more and more susceptible and have less and less reserve. This is based on observation of certain aspects of the anatomic lesion. In many muscle cells not showing contraction band formation or myofibrillar degeneration, large numbers of mitochondria have been moved away from the contractile fibrils and are pooled in other areas of the cell. This has to affect the metabolism of that cell. There are also other lesions such as swelling of membrane, increase in the amount of fluid inside the cells, and changes in the myofibrils themselves, that would seem to indicate that the affected cells are less capable of contracting and carrying their own weight which will put more of a strain on those cells which are left. In other words, each time there is a superimposition of a lesion-producing stress, there is less reserve left. Over a lifetime - if there is a myocardiopathy produced in some pilots - and if he continues to fly, and it resolves as it does resolve in the swine, there is going to be an increase in connective tissue deposition in the endocardium of the heart.

CLARKE: Dr. Stone, will you comment to this question, please?

STONE: (United States) Yes. Let me back up for just one second and make a general comment. Several years ago I had the honor to attend a symposium concerning the use of miniature swine in biometric research. The collection of scientists at that particular meeting also asked the question, "Is man more like a pig or is pig more like a man?" So, the utility at least in a non-flying environment of minature swine as a possible analog particularly of the heart for man has been relatively well substantiated. Now as far as in the hazard of the environment in which a pilot would be placed in high performance aircraft, I think that is an open question at this point in time. However, I would think in direct answer to the questions posed that there is no doubt in the animals we have used, some subclinical damage has been done to the myocardium. We have been able to follow this over a period of days in the absence of measured changes that would be of physiological consequence. I feel that this means that our techniques for measuring physiological changes in the intact organ are rather gross. I think that all of us should appreciate that so when anyone states there are no physiological changes that are evident, what he is really referring to is that he can measure. Therefore, I would suspect and from data conclude that repeated exposure to high levels of acceleration would produce some sort of myocardial deficit. How or whether this would show up in man after years of flying ACM is conjecture at this point. I think that short intervals between repeated exposures - say days - may not completely ameliorate the situation. We saw in the animals that they required up to 14 days for many of the parameters to return toward normal. We could conclude that any reexposure up to 14 days may exaggerate any degeneration that was initially observed. The overall role of the coronary circulation, I think, is the real key here and how it performs in man as opposed to the swine. There are several parallels that can be drawn about depolarization and repolarization of the myocardium in both animals and man. Irregular mobility of ions across cardiac muscle cells, irregular dispersion of activation by the autonomic nervous systems - specifically the sympathetics - I think, would cause an increased number of problems in both pilots and animals.

CLARKE: (United States) Dr. Hamlin's paper today reminded me of one of the founders of microcirculation back in the old days. It was Dr. Krogh, from this part of the world I believe, who made probably one of the most profound statements about the cardiovascular system that I've ever heard and his one-liner was, "The blood goes where it's needed." And if you think about that in the context of Dr. Hamlin's paper I think that's pretty well the case. But I wonder, Dr. Hamlin, if you see in this pattern of cardiovascular change that involves not only the coronaries but the other circulatory factors anything that would tend to cause concern for the questions that we posed here.

HAMLIN: (United States) I ask myself the question trying to come to grips with it, being basically a chicken - if I weren't a chicken, would I fly high sustained G? I try to look at the evidence and it really is not overwhelming that it's hazardous. I hear from my colleague on the right (Dr. Voge) that even the arrhythmias may not be present in a situation that might simulate more accurately what one sees while flying a jet aircraft - that is with a gigantic radius of curvature. Whereas, my colleague on the left (Dr. Stone) and I find arrhythmias in animals that are subjected to sustained G on a centrifuge, with a smaller radius of curvature. I see subendocardial hemorrhages in swine that I don't think would bother our Food and Drug Administration terribly, particularly in the light of the fact that they go away. I hear my friend from Norway (Dr. Sem-Jacobsen) suggest that maybe alcohol and all the stresses and strains that one would think would cause hazards in flight, don't in fact cause hazards. And I wonder if maybe most or all of this could be a manifestation of input from the hypothalamus to the autonomic nervous system and that many of the arrhythmias and indeed some of the potent adrenergic input to the heart may take their origin in the cerebrum and get to the heart by the thalamus and hypothalamus? I wonder if some of the most important factors, i.e. the psychological factors, haven't really been adequately considered. We possibly have not come to grips with some of the most potent of the factors that determine the input to the heart.

LEVERETT: (United States) I just wanted to make sure that we all understood now what Dr. Hamlin just said and what Dr. Stone just said. One states that in looking at the overall picture it doesn't worry him too much about pilots pulling high sustained G or ACM type G while the other one, who sees the swine hemorrhage everyday, is a little concerned about it. The question of the vectorcardiographic changes that Dr. Gillingham discussed were brought out in the one study by Dr. Forlini. As Dr. Gillingham stated, if one had visually observed the VCGs at that time they would have not been considered pathologic and the shifts in the T-loop would not have been considered pathologic. However, these changes occurred and the shifts were in a direction that may indicate myocardial hypoxia and this plus the endocardial hemorrhage in the pig that Dr. Burton had found meant that we should at least be aware

that it occurs and we should then look at all of our human subjects who were carried to levels above 7 G.

CLARKE: (United States)

Dr. Lisher from the United Kingdom represents perhaps a slightly different view point in as much as he brings work from a different laboratory. Although your paper is oriented in another area, I wonder if you would express your opinion here regarding the pathology, please.

LISHER: (United Kingdom)

I feel hardly qualified to speak but I do tend to agree with what Dr. Gillingham said, in that I think as soon as we find that aircraft like the F-15 and F-16 are introduced into service your figure of intensity of 4 G will gradually creep further and further up. And that fighter pilots being fighter pilots will probably use the aircraft performance more and more until we find ourselves in a situation where perhaps they are at 7 G for 60 sec with excursions to whatever the airplane will allow them to pull. I think this is a completely new question. I believe it is probably true to say that in the dogfight situation, fighting against a comparable airplane, it seems duration at G is the winning point.

CLARKE: (United States)

I think this is a very interesting and useful observation that both you and Dr. Gillingham have made. The basis for my statement of these figures was essentially from the ground based simulator work at McDonnell Aircraft Corporation where the pilots were not necessarily limited by the physiological effects of G but rather they could turn as tightly as they wanted to with only the G bladder pressing gently against their tummy. In these simulations, they tend to still work in spurts and tend to get on target with high bursts of acceleration and then back off and fire. That may represent a lack of training on their part in terms of their ability to take advantage of the performance of the aircraft, so I suspect your two conclusions are what really will occur in an equivalency air battle.

We heard this morning from Dr. Voge from the U.S. Navy some of the biochemical changes that were associated with her experiments at reasonably low levels of acceleration. Would you like to comment on the significance of these changes vis a vis the operational situation?

VOGE: (United States)

We actually do not know what the physiological risk to a tactical fighter pilot will be. I am not sure how much we can infer from the miniature swine studies. Another thing we must consider is that in a real life situation the fighter pilot isn't just subjected to acceleration. He's subjected to such things as buffet, heat, varying light condition and many other environmental stresses in addition to acceleration. If he has a single exposure, or several per day for several days, fatigue may be a factor. As far as G tolerance is concerned, we get an increase in G tolerance in our subjects if they're trained. If the trained subjects don't practice for two or three weeks or a month, they lose this ability they've gained. And the training that is required to enable them to be able to withstand higher forces is not known by our group at Warminster yet. Of course, we still have difficulty with the problem of 100% oxygen - that's definitely a problem as far as the U.S. Navy is concerned.

CLARKE: (United States)

Dr. MacKenzie mentioned the variability of the individual as a function of age and condition and Dr. Sem-Jacobsen has pointed out inherent variabilities in performance of fighter pilot populations and I believe these are perhaps somewhat related, at least in indicating that there is a source of variability that enters in answering this kind of simplistic question. Would you like to comment as to your feelings, Doctor?

SEM-JACOBSEN: (Norway)

Yes. I would like to make just a few comments. From my study we learned that you can have a number of fighter pilots that only tolerate 3 G and we have a very large group of pilots that very well tolerate 9 G for a long period of time. We have, however, one concern when you ask the question about high G exposure and repeated G exposure. When we are pulling excess G, 9 G or more, the blood pressure in the left ventricle may be up to 400 millimeters of mercury. The heart, surrounded by the pericardium, acting as a very good anti G suit protects the heart muscle from bursting. However, Dr. Franks in Canada found that a number of pilots' hearts had ruptured at autopsy. He raised the question "is this rupture due to repeated G abuse to the point where the pericardium failed and no longer acts as an effective anti G unit?" This same thing occurs in the knee joint and leads to a sloppy knee capsule. Dr. Franks also reported massive heart muscle hemorrhage in jet fighter pilots during aircraft crash investigations. Is this due to stretching the pericardium in these instances? I think this is a thing that should be looked into in animal work before we can really say whether repeated G exposure is hazardous or not. (Note: One would have to rule out effects of crash forces in producing cardiac lesions to infer that manuevering accelerations were causally related.)

CLARKE:
(United States)

Thank you very much. I will try to summarize a few of the more important points on what we know and don't know about the question I posed. I believe it is fair to say that we have not done the human experiments that let us conclude that frequent exposures over a short period of time produce no effects. I believe it is also fair to say such a conclusion is a conjecture based on the best information we have and, do not misunderstand, I think that is the kind of conjecture we are asking for today. We are asking the best opinion based on the knowledge that we have but it is important to say that the key experiments have not been done. And, of course, we do not have experiments that clearly establish in man what the effects of exposures over a career to this environment will produce. Dr. Leverett pointed out that it is important to keep in mind that we have never seen these kinds of subendocardial hemorrhages or other cardiovascular changes in man that can be clearly and uniquely related to having been exposed to a high air combat maneuvering type of environment. We have seen these kinds of lesions in experimental animals and I do not believe we have mentioned it today but we have seen them in multiple species of experimental animals. I refer to at least one series of experiments that Henry and Gauer did back in the early 50's with the goat. I am not sure whether there are studies in other species or not, but we do have at least some indication of these kinds of lesions occur in other animals. One of the things that makes this kind of question about the permanent effect on skeletal muscle of this kind of damage we would have a significantly different interpretation as to its potential effect than we do being concerned about cardiac muscle and the obvious vital role that it plays. There is another area of concern that we really have not addressed very well that has been identified by other workers. I refer to Dr. Earl Wood from the Mayo Clinic who attended the Cocoa Beach session. He expressed concern about the possible mechanical failure of lung tissue as well as damage, to the cardiac muscle per se as being one of the factors. This potential may be greater in the reclining seat where the levels of acceleration are not limited by loss of vision, but are limited by other factors. I guess we haven't really nor did we expect to come to a crisp, clear yes or no.

And quite a bit of the remaining discussion has centered itself around that set of conjectures that Dr. Gillingham made expressing the reservations that we have about these conclusions. In many cases I hope expressing clearly the need for additional experimental data and the difficulty of the problem will provide some indication to the military committee and to the operational community of the seriousness of the problem to be addressed. In the interest of time, we should probably press onto our next question which I believe can probably be answered a little bit more succinctly.

The second question submitted to the speakers was: "WHAT ARE THE PHYSIOLOGICAL FACTORS THAT PROVIDE FOR PRACTICAL PERFORMANCE ENHANCEMENT DURING HIGH G MANEUVERS AND WHAT ARE THE LIMITS OF PERFORMANCE ENHANCEMENT THAT THEY PROVIDE?" Here, to some extent, we are talking about statements of the obvious in terms of identifying the factors, but perhaps the speakers can address themselves a bit to the quantitative aspects of this question -- "How much protection is provided by each of the mechanisms?" The straining maneuvers that Dr. Leverett described as training procedures are certainly important factors. We've introduced a new factor in our thinking here with the possible payoff of positive pressure breathing. We will absolve talking about body position including back angle in our discussion. As we address this question I wonder if we might start to define the operational break point where there is a clear and justifiable advantage to cockpit redesign that is necessary to take advantage of the tilt-back seat position. Where in the range between 7-10 G, would you advise the designer that he clearly needs to go to the tilt-back seat or to some other positional change that gives a pilot the postural advantage to perform well in the acceleration environment? Here again, we might let each one express briefly their idea on this. Dr. Leverett, would you like to start?

LEVERETT:
(United States)

When we first addressed the question of high, sustained $+G_z$, we started at $+6G_z$ for 45 seconds and increased the intensity to $+9G_z$ for 45 seconds. Exposures up to levels like that are similar to the exposures that the McDonnell fixed-base simulator indicates that an F-15 pilot would utilize against a threat aircraft. He pulls high G for brief periods of time to get into a favorable advantage. We have asked the question that is obvious, i.e., "How will a pilot use high sustained $+G_z$ and in what type engagement scenario will it be used?" In the Lufbery circle type maneuver, one plane attempting to get on the tail of another may entail high sustained $+G_z$ but it may not exist in a real operational situation. The worst case would be an opposite direction wing-to-wing engagement between two aircraft in which each aircraft would make 180° turns in order to gain some advantage. They might be pulling high G for 15-20 seconds or so. But if we showed in our 9G study that at least a pilot can maintain vision at 9G for 45 seconds, then the next question is, "Do you want to go to higher G levels and if so, when should a tilt-back seat be incorporated in cockpit design and at what level can G become a debilitating problem?" In watching how hard subjects worked at $+9G_z$, I would say you better start tilting them a little bit early, earlier if

they are really going to pull high sustained G. If they are going to pull peaks at 9G, I don't think there is a problem. If they are going to peak to 12G, then you had better start tilting them at 9G. In the +8-10G_z range, a pilot can lose consciousness very easily if he does not pace his breathing and straining maneuver. We did have one pilot go unconscious on us at 8G. It is my opinion that one should begin tilting the pilot back at levels above +8Gz. We saw from our studies with the RAF and from Dr. John Burns' (USAFSAM) experience that you must tilt the pilot back further than 45° to gain a G advantage. When you talk about tilting a pilot back, you should be prepared for some initial negative feeling from him when he does not want to go back to 65° to gain this added protection. You can expect the operational community to be negative to a tilt-back of this magnitude (65° to gain added protection).

CLARKE:
(United States)

In the question of optimum angle of tilt of the seat, we do have the angle of attack of the aircraft working for us in providing an optimum tilt of the seat. It can add 10-15° of effective back angle in some circumstances. Dr. Lisher, would you express your feelings and those of your colleagues, please?

LISHER:
(United Kingdom)

I must admit I completely agree with what Dr. Leverett said. I think the real factor is that we are going to expect pilots to pull higher G and perform at these levels. We are going to expect them to not just fly that airplane - we are going to expect them to fight with that airplane also. In such a situation, I think the mere maintenance of an adequate visual field by attention consuming M^1 maneuvers or something like this is putting an undue strain on the pilots. I also feel that it is in this current generation of aircraft (12 G capability) where we should consider some form of reclining seat or a tilt-back seat.

CLARKE:
(United States)

Would you comment a little bit more on or perhaps just summarize what you said this morning on your feelings about the operational utility of positive pressure breathing (PPB) for enhancing G tolerance?

LISHER:
(United Kingdom)

Yes. As a side issue of our main experiment we did a small experiment on the effect of positive pressure breathing on G tolerance on people in a conventional seat. We used a gradient of 5 torr per G for our breathing pressure. Relaxed, unprotected subjects had a blackout threshold of 3.5 G which these same subjects using an anti-G suit had a blackout level of 4.75 G with positive pressure breathing this level increased to 5.5 G, thus an increase of about .75 G protection by PPB alone. In this seated upright situation, you have to work at expiring against continuous positive pressure breathing which is not required in a reclined seat where it is all quite simple. During our reclining seat experiment we were limited to about 40 torr breathing pressure by the oxygen regulator which we were using. One question we have yet to address is "if we go to higher than 8 G, will people be able to tolerate PPB at 45-50 torr, for example?"

CLARKE:
(United States)

Thanks very much, Dr. Lisher. In considering the tilt-back seat or in taking advantage of reclining the upper torso, some of the limitations that have already been addressed in practical cockpit design and they have to do with the effect that this has with placing the man inside the aircraft in terms of geometry. Also the human engineering people have given considerable thought to the question that was posed earlier this morning of how one goes about seeing out of the aircraft while in the reclined position. The people at the Aviation Medical Acceleration Laboratory at Warminster, Pennsylvania have spent some time thinking about this and have developed some innovative approaches. I wonder, Dr. Voge, if you would address that area for a few minutes?

VOGE:
(United States)

I think it is well known that the M-1 maneuver adds about 1½ to 2 G's to tolerance. On top of that, a G suit in either the reclining position or in the upright position adds another 1½ to 2 G improvement in tolerance. When we "kick" them up to high G's in the upright position using the M-1 maneuver and the G-suit, they become extremely fatigued. Even in the reclining position, we have the problem of pressure on the chest which the positive pressure breathing technique may take care of. We found that the seat-back angle would probably better than double the G tolerance. The problem as far as head mobility is concerned with our type of seat - the kind we are evaluating at the present - the head remains stationary. What moves are the pelvis and legs. They are elevated to about 59°; the back is 75°; the upper legs 59°; and the lower legs to about 118°. We also studied the problem of the cockpit size with respect to leg position. This last year, we completed a series of tests in which the legs dropped straight down in order to save cockpit space. We found no decrease in G tolerance. In fact, we had a slight increase in G tolerance although this was not significant. We already have a 12 G aircraft - the F-14 Tomcat will be 12 G½s - our pilots cannot take 12 G's. So, right now we are ready for a tilt-back seat with our existing weapon system. Perhaps, as far as fatigue is concerned, the pilots after about 7-7½ G's become extremely fatigued in the upright position performing their

M-1 straining maneuver and, of course, they may have a certain amount of tunnel vision. I think that at 7-7½ G's we should start thinking about having a tilt-back seat. We have had several fighter pilots ride our reclined system and they consider it to be quite comfortable and they are very, very pleased with it - very enthusiastic.

CLARKE: (United States)

Thank you. The question we really cannot address today because of a limitation of time would be to ask ourselves, "Is there a practical redesign of the cockpit of an existing aircraft that would allow some productive compromise between a no tilt seat cockpit and one in which the aircraft was specifically designed and constructed with a tilt back seat?" We know one must tilt back quite a ways (45°) before you begin to get any real benefit. To review briefly the historical development of the tilt back seat concept we know von Diringshofen explored the flop-back seat back in the 1940's; the U.S. Air Force examined it again in the mid 50's. We re-examined these positions to find the optimal situation for space-flight accelerations during the late 50's, and now we are looking at it yet again for use in fighter attack aircraft. The new opportunities that we have in control and display technology may well allow the pilot to assume this position and not only maintain all the advantages that he has in control and display now, but in some cases to actually enhance his capability. I wonder if anyone else on the panel would like to comment on this question before we move along to the remaining questions.

SEM-JACOBSEN:

I just have a very brief comment. If we want a pilot in an aircraft, it is because we want to use all his knowledge, training and experience. Therefore, I feel that we should really start placing him in a reclined, protected as early (at 5 or 6 G) using PPB in order that he is not fatigued. So, I think we should move right ahead in this development.

CLARKE: (United States)

Dr. Gillingham, did you have a comment?

GILLINGHAM: (United States)

Yes. We were discussing the physiologic factors that provide for practical performance enhancement, and I would just like to review some of the things that have been said. There are two main physiological factors: 1) arterial blood pressure and 2) oxygen saturation. Blood pressure can be increased by 4 factors that we have discussed: 1) the M-a maneuver or L-1 straining maneuver, 2) a tilt-back seat, 3) positive pressure breathing, 4) combinations of these. The positive pressure breathing has been covered by my colleague from the RAF (Dr. Lischer). The M-1 maneuver has the disadvantage that it causes a tremendous decrement in performance. The seat angle does not have that disadvantage in that one can perform very readily at high G in a tilt-back seat. It does have a possible disadvantage in that we believe that the lung is more sensitive to G forces in that direction. Perhaps not more sensitive but that the fact that one can go to higher g's results in the ventilation/perfusion abnormality and resultant desaturation. Now, one can do a lot of performing while he is desaturating between 97% and 60%, but when one's blood pressure drops down below intraocular pressure or below, say 30 millimeters of mercury, one cannot do any performing. I suspect if one had to choose between the various methods of increasing G tolerance, one would definitely take the tilt-back seat because it gives you the increased performance with only a slight physiologic cost. The other problem then comes up - do we design a seat or do we design an aircraft that can accommodate a tilt-back seat. It seems to me the time has come to stop asking the question whether we can put a tilt-back seat in an airplane as an afterthought. If we are going to design any more manned airplanes, it is hard for me to believe that we cannot design an airplane that will accommodate a man in a 50-55 degree seat which, with his angle of attack, will put him into a 65 degree seat.

CLARKE: (United States)

Thank you very much for the comments on this question. I would like to take the next question now in the area of the "USEFULNESS OF CENTRIFUGES AS TRAINING DEVICES" and inasmuch as the Italian authors are not here, we might ask Dr. Leverett to comment on this question and ask for any other feelings that might be expressed by the panel. Just by way of introduction, there is one aspect of the training program at the School of Aerospace Medicine in the United States that Dr. Leverett only touched on very briefly, on which I might comment to a little more. A useful adjunct to the pilot training is the centrifuge training of all of our Physiological Training Officers who go to the field as well as all of the Flight Surgeons who are going to be supporting fighter pilots in the tactical forces. High G training is provided to them at the time they go through their other formal course of training at the USAF School of Aerospace Medicine. This is gradually giving us a cadre of personal equipment people, physiological training officers, physiological training technicians, and flight surgeons in the field who all have first hand experience with high G and who are able to provide current information to pilots during the course of their normal refresher training in the field. The thing, perhaps, that would be most useful to our report to the AGARD Military Committee in this area would be to state a) what kind of training regimes you would believe to be optimal,

b) would repeated training be required during the course of the fighter pilots career and c) any other factors you might think be appropriate, Sid.

LEVERETT: (United States)

Some of this is going to be anecdotal. When we first started training the highly experienced F4E pilots, it goes without saying that you do not tell him his straining maneuver during high G is being improperly performed. It was best to present the material in a lecture foremat early in the morning and then put the first one or two pilots on the centrifuge. They will usually not do a straining maneuver properly because they are not experienced at high sustained $+G_z$ and it is difficult to pace your breathing until you practice at G for a bit. The training program we developed at SAM turned out to be good and was universally accepted by them (the pilots). We modified the original G exposure slightly in order to put in another run or two. These included high sustained G plus a run stimulating an ACM environment with two peaks of 7G over a time period of 94 records. We can also fool them on our centrifuge and make them believe that they are controlling the centrifuge. I would strongly recommend that new centrifuges being built for high G training be capable of being controlled by the pilot receiving the training. The closed-loop centrifuges like the U.S. Navy centrifuge at Warminster or like the German Air Force centrifuge at Furstenfeldbruck are closed-loop and thus the man can modify the G level himself. I think it is important that they feel they are controlling the centrifuge. Otherwise they are going to view the training as some other passive simulator which the medical community has imposed on them and I don't believe they will accept it as well. We also give them a tracking/shooting task to perform in order to maintain their attention during the G exposure. Everything is designed to make the training as realistic as possible. They wear their own flight clothing including boots, gloves, helmet, oxygen mask, flight suit and anti-G suit. The tracking task makes them compete against each other and it becomes a realistic affair as they observe their fellow pilots perform on closed-circuit television. The closed-circuit television is almost a necessity in a training program of this type. They observe themselves, their fellow pilots and can be criticized during re-runs of the videotape by the physicians and physiologists who monitored their runs. Put a voice track on the videotape and they can hear how bad their straining maneuver was. We have found universally that all pilots can repeat their "bad" centrifuge run and make corrections to such an extent that you cannot get him up to his upper tolerance limit at the planned exposure levels.

Dr. Ewing raised the question this morning about the effectiveness of a G-suit. Most anti-G suits are advertised as protecting a pilot about 1.5 G. However, when the suit it used with a properly performed straining maneuver then we do not know what the upper limit is.

THE M-1 AND L-1 STRAINING MANEUVERS

M-1 Maneuver -- Pilots commonly refer to the M-1 maneuver as the "grunt" maneuver since it approximates the physical effort required to lift a heavy weight. The m-1 maneuver consists of pulling the head down between the shoulders, slowly and forcefully exhaling through a partially closed glottis, and simultaneously tensing all skeletal muscles. Pulling the head downward gives some degree of postural protection (shortens the vertical head-heart distance); intrathoracic pressure is increased by strong muscular expiratory efforts against a partially closed glottis; and the contraction of abdominal and peripheral muscles raises the diaphraghm and externally compresses capacitance vessels. For long-duration G exposures, the maneuver must be repeated every 4 to 5 seconds. When properly executed, the exhalation phase of the M-1 results in an intrathoracic pressure of 50 to 100 mm Hg, which raises the arterial blood pressure at head level and thereby increases +G tolerance at least 1.5 G. The inspiratory phase of the M-1 maneuver must be a fast "gasp", to be followed immediately by the exhalation phase, since mean blood pressure falls to approximately zero during inspriation.

L-1 Maneuver -- The L-1 maneuver is similar to the M-1 maneuver except the aircrew member forcefully attempts to exhale against a completely closed glottis while tensing all skeletal muscles. Using either maneuver the pilot obtains equal protection, i.e., 1.5 G greater than relaxed blackout level with or without the anti-G suit. In a 1972 study, subjects wearing anti-G suits and performing either the M-1 or L-1 straining maneuver were able to maintain adequate vision during a centrifuge exposure of $+9G_z$ for 45 seconds. Higher and longer runs have not been attempted. However, it is important to note (a word of caution) that forcefully exhaling against a closed glottis without vigorous skeletal muscular tensing (Valsalva maneuver) can reduce $+G_z$ tolerance and lead to an episode of unconsciousness at extremely low G levels. Therefore, instruction and training on the proper method of performing these straining maneuvers is essential. (Source: Gillingham, K. K. and R. W. Krutz, USAFSAM AR 10-74, Brooks AFB, TX 78235.)

LEVERETT: (United States)

We have gone up to 9 G for 45 seconds with the suit and with straining but we really cannot tell the pilots or you what the upper limit of $+G_z$ tolerance is at the present time even in the upright seat. If we can show a pilot how they can

improve their tolerance and ability to perform while somebody is shooting at them or while they are trying to acquire a target, then I think we have done our job with the centrifuge training.

CLARKE: (United States) From what you said, the other conclusion that one can make is the high G training takes a relatively short time out of a busy fighter pilot's schedule in order to improve tolerance and confidence at high G. Also, I guess you think it is not required to have repeat visits to do this so it could be a one time session. Where in the training or career of a tactical fighter pilot would you recommend that this centrifuge training would occur? Is it during his primary training or is it after he has had some experience?

LEVERETT: (United States) That is a good question. I am glad you mentioned it. It is not during primary training. They get a little bit of this in physiological training but before a man graduates from pilot training, he feels he is probably the greatest flyer in the world. He does not really realize how much he needs to learn until he graduates and begin flying ACM. Then when he gets a few hours as a fighter pilot he really realizes what he does not know and what he did not learn. Universally, our experienced fighter pilots who came in for the training felt that it was beneficial. I think that they felt they would not have gained as much out of it if it had occurred in their student pilot training period.

CLARKE: (United States) Thank you very much. I would like to move to another question and since our time is running short, if it is agreeable to the panel to take just one answer for this. I have been told that there are some experiments underway now looking at the question of whether or not physical conditioning can enhance a pilot's ability to perform under these very high levels of acceleration. And here again, if I may, I turn to Dr. Leverett, who is the individual who reported these experiments in progress and I ask if he would be willing to make a brief comment on where the studies are now and what conclusions we might tentatively draw.

LEVERETT: (United States) We do have a study going on now. We are looking at the difference in tolerance between subjects who are in a weight lifting program compared with those in a running program. Many of you are presented the same question, i.e., "what is the best physical conditioning regime to improve and maintain G tolerance?" By the 15th of July we will have the data collected and expect an answer to this question shortly thereafter. We are going to use a little bit different method of determining tolerance rather than just using loss of vision during a single exposure. We are exposing the subjects to repeated $+4.5G_z$/15 seconds then $+7G_z$/15 seconds G profiles, never coming back to 1 G until the subject stops the run due to fatigue. At the present time before I came over here last week, we had some of the subjects in our panel who were lasting up to 6 minutes. We believe this is a more realistic profile to determine an endpoint to G similar to the pilot on a mission engaging until he is fatigued.

CLARKE: (United States) Thank you. The next of our questions is not strictly related to high G maneuvering and the kinds of pathophysiological effects that we have talked about today. But the panel of speakers that we have here are probably as well qualified to address the question as any group we could get together. And I would like to pose this question to them. I am told by tactical people and by operational flight surgeons that there is some operational concern for the individual who is not doing air-to-ground ordinance delivery in which during the course of one or two days he might be exposed to lower levels of acceleration down in the 3 or 4 or 5 G range repeatedly time after time during the course of these kinds of missions. There is perhaps some reason to assume - based on fairly limited analysis of operational accident data - that these people when they are exposed to this environment and otherwise suffering from the fatigue of combat are more apt to have accidents than they are if they are not so severly stressed in terms of multiple exposures. This led to posing our last question then which is, "WHAT ARE THE PRACTICAL SAFE LIMITS IN TERMS OF PILOT PERFORMANCE AND FATIGUE TO REPEATED LOW LEVEL ACCELERATION MISSIONS DURING THE COURSE OF A SINGLE DAY?" Dr. Lisher, I know the people at Farnborough have given considerable thought to this operational environment in times gone by. I wonder, perhaps, if you could start the discussion on that?

LISHER: (United Kingdom) Yes, I think that really this is one of those questions to which the experiment has not yet been done. And, I think that there is obviously a situation which occurs when another 30 seconds at 3 G is going to be too much for one particular pilot in that he is going to feel really tired and fatigued after this. And, as I say I really do not think the experiment has been done. It is obviously a matter of trying somehow to quantify fatigue and with all the subjectivity that that involves. I do not think I can say anything more.

CLARKE: (United States) Thank you very much. Dr. Gillingham, would you comment on it, please?

GILLINGHAM: Regarding the 3 G level, I am not impressed with its stress, really. Based upon the TAC pilots that I have talked to and with my own experience in acrobatic flying, I do not believe that 3 G puts much of a stress on the individual. And, I that one can tolerate this repeatedly for many hours. One thing that I have heard, though, from the field is reports of several accidents that have been caused in bombing missions. And I think what is happening there is that these pilots are pulling more G than what we think they are. I do not believe these are 3 G maneuvers. I think we are talking now about 7 plus G maneuvers during these bomb releases. And, now you are getting into the problem where there may be chronic fatigue that can lead into a sudden loss of consciousness. Medically, I have been impressed by the fact that during a loss of consciousness on the USAFSAM centrifuge (about one per month) the subject has no recollection of this episode. His head suddenly drops down, the centrifuge is stopped, the door is open, people are in the centrifuge gondola, and he is asking "What happened? Where am I? Why? When are you going to start?" There is a little bit of post run depression and amnesia and I think this may be one of the reasons we are getting into trouble during bombing runs that are repeatedly conducted.

CLARKE: (United States) This is similar to the experience you have had, Dr. Sem-Jacobsen, in some of your studies, I believe, is it not?

SEM-JACOBSEN: (Norway) Yes, I would like to mention that the EEG might be helpful in this evaluation.

CLARKE: (United States) Thank you very much, sir. Our time is just about run out. So, perhaps we should conclude our session for the day. I would like personally to thank each of the speakers not only for their very fine presentations, but also for agreeing to be put on the spot in this round table discussion. I hope this has been productive to the audience. I know it has been extremely interesting and informative to me. Thank you in the audience for your many provocative questions during the day. Your participation helped make this a useful and interesting session. Our session is adjourned until tomorrow morning. Thank you.

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14. Abstract

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